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ALPHA CONTROL AND ITS MEDIATING
EFFECTS ON PAIN AND ANXIETY

Robert R. Dunne

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Monterey, California



THESIS

ALPHA CONTROL AND ITS MEDIATING
EFFECTS ON PAIN AND ANXIETY

by

Robert R. Dunne

and

Scott B. Dudley

March 1976

Thesis Advisor:

T.A. Wyatt

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Alpha Control and Its Mediating
Effects on Pain and Anxiety

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I. INTRODUCTION

A. BIOFEEDBACK

1. Definition

"Feedback", as defined by mathematician Norbert Weiner is "a method of controlling a system by reinserting into it the results of its past performance" [Karlin and Andrews, 1972]. The senses of sight, hearing, taste, feel, and smell provide us with the necessary feedback to react and exist in our environment. "Bio" is a form of the Greek word "Bios" meaning life or living organisms. "Biofeedback" thus refers to the process by which individuals are given instant information about their mind or body using electronic instruments or monitors.

Traditionally, scientists believed there were two distinct sorts of body activity, somatic (voluntary) and autonomic (involuntary). Speech, arm and leg movements, as well as the arching of an eyebrow were among the functions that a person could will for himself. But activities such as brain-wave patterns, gastric secretions, fluctuations in blood pressure, heart rate and skin temperature did not seem to fall within man's conscious control.

With the advent of biofeedback techniques in the 1960's, many scientists now think that if a person can learn about the internal rhythm of a particular body process through electronic feedback, then he can also learn to exert a degree

of control over the process in question. Biofeedback learning occurs as a person becomes able to perceive the minute internal happenings of his body and mind, and learns to "feel" how to control those events. Biofeedback is analogous to a mirror, in that, like a mirror, it gives immediate feedback about ourselves. Moreover, just as a mirror can help us exert external control over our voluntary functions, likewise, biofeedback can help us exert internal control over our involuntary functions.

The principle of biological feedback is not new of itself. An everyday example of feedback is seen in the eye-hand coordination, where both visual and muscle information feeds back to the central nervous system at every point in the series of movements comprising the act of reaching for an object. This feedback makes possible precise control at every moment. The new elements added by biofeedback are "the external detection of physiological activity, and in many cases, augmentation of the feedback by the addition of sensory channels" [Kamiya, 1971].

2. Classical Versus Operant Conditioning

Throughout the literature on learning theory there are references made to "classical" as opposed to "operant" or "instrumental" conditioning.

In classical conditioning, an unconditioned stimulus, known before to be a reliable elicitor of a specific response, is presented during or shortly after the presentation of

another stimulus, the conditioned stimulus. The conditioned stimulus (such as a bell) does not normally elicit the response of the unconditioned stimulus. The conditioned and unconditioned stimulus pairs are presented repeatedly, regardless of variations in response. Eventually, if the conditioning is successful, the conditioned stimulus begins to elicit the response that previously could only be elicited by the unconditioned stimulus.

In operant conditioning, a rewarding (reinforcing) stimulus which the subject would normally expend effort to obtain is given immediately after the subject makes a response pre-selected by the experimenter. This results in the subject making the response more frequently and obtaining rewards as he proceeds.

The two types of procedures differ in one major respect -- the relationship of the stimulus to the response. In classical conditioning the reinforcing stimulus is given with the conditioned stimulus regardless of the subject's response. In operant conditioning the reinforcement is given contingent upon the occurrence of a pre-selected response. The reinforcer also does not by itself provoke the response to be conditioned [Kamiya, 1971].

Miller [1971] has pointed out that since ancient times the voluntary responses of the skeletal muscles were considered to be superior, and the involuntary glandular and visceral responses to be inferior.

Similarly, there was a distinction made between a lower form of learning called classical conditioning, which was thought to be involuntary, and a superior form of learning called operant conditioning, thought to be responsible for voluntary behavior.

Historically, these distinctions coalesced into the strong traditional belief that the superior type of operant learning involved in voluntary behavior is possible only for skeletal responses mediated by the cerebrospinal nervous system. Conversely, the inferior classical conditioning is the only learning possible for involuntary visceral responses mediated by the autonomic nervous system [Miller, 1971].

However, Miller [1971] did not believe that operant learning and classical conditioning were two basically different phenomenon, but rather were "different manifestations of the same phenomenon under different conditions." Failing to see any clear cut dichotomy between the two, he assumed that there was only one kind of learning. His assumption demanded that operant training procedures be able to produce the learning of any visceral responses that could likewise be acquired via classical conditioning.

3. Biofeedback Training

The biofeedback training method is relatively simple and is based on the same principle as operant conditioning. The only difference is that the biofeedback studies require the performance to be an internal physiological change,

whereas operant conditioning as originally developed required an external act that "operated" on the external environment or at least was an externally observable movement [Kamiya, 1971].

The training technique as Kamiya [1971] outlines it has three requirements:

(a) The physiological function to be controlled must be continuously monitored and the monitor must be able to detect moment-to-moment changes.

(b) The changes in the physiological measure must be manifested immediately to the subject.

(c) The subject must be motivated to learn.

4. Biofeedback Research in General

Miller and Di Cara [1967], in challenge to the traditional view that visceral responses can be modified only by classical conditioning, showed clearly that curarized rats could be trained to either increase or decrease their heart rates by rewarding the desired change (operant conditioning). They emphasized the fact that these changes were obtained in rats with complete paralysis of the skeletal musculature and thereby ruled out any possibility that this visceral response was mediated by instrumentally learned, overt skeletal responses.

Shapiro et. al. [1971] gave 20 normal male subjects feedback of their own systolic pressure. Half the subjects were operantly reinforced for increasing their blood pressure, while the other 10 were reinforced for decreasing their

pressure. The results of their study indicated that systolic blood pressure can be modified by the use of external feedback and operant reinforcement. Their apparatus and techniques suggested a possible approach to modification of blood pressure in hypertensive patients.

Green, et. al. [1969] used an electromyographic (EMG) feedback technique as a method to achieve deep relaxation of striated muscles. They pointed out that a general problem with traditional relaxation procedures is that it often takes days, or weeks, before a subject or patient can relax to a satisfactory degree. Through the use of their simple EMG feedback method, subjects were able to voluntarily achieve extremely low tension levels, tending toward zero, in a few minutes. For example, 7 out of 21 subjects were able to achieve intermittent neuromuscular silence within 20 minutes and maintain it for the duration of an experiment of 30 minutes or more. Before using the feedback system, they were unable to approach the zero level of tension "quickly" in any subject.

Taub [1971] hypothesized that a rapid-reacting temperature probe would provide a simple, accurate, and easily set-up means of observing and controlling regional blood flow. Operating on this hypothesis, Taub and his staff developed techniques for enabling most humans to establish rapid operant control of their own skin temperature, when provided with immediate feedback information concerning variations in local skin temperature. Furthermore, retention.

of the task was found to be "virtually perfect" over an interval of 4 to 5 months. It was also found that, after sufficient training, auto-regulation of skin temperature was as good without feedback as with feedback.

These four studies have been highlighted just to offer some substantiation to Miller's [1971] claim that operant training procedures are capable of modifying and controlling autonomic responses.

B. ALPHA FEEDBACK

1. Definition

Brain wave feedback is a form of biofeedback training in which a person learns to control his cerebral electrical activity. Again, alpha feedback is a form of brainwave feedback wherein operant conditioning procedures are utilized to reinforce the particular brain wave activity called alpha.

2. The Human Brain

The brain is an organ which is composed of many parts, and of millions of cells within those parts. The primary cells that compose the brain are known as "neurons". While a neuron in the human brain may be as small as a few thousandths of an inch, a neuron carrying information from the extremities of the body to the brain might be 2 or 3 feet in length. A single neuron is composed of three parts: the axon, the neuron body, and the dendrite. The dendrites are fibrils at the receiving end of the neuron and the neuron may have more than one of these. The traditional view

advocates that there is only one axon, the fibril that conducts away from the cell-body toward the next cell, but it usually has a number of branches or collaterals. The dendrite receives an electrical impulse through its tips and transfers this pulse to the neuron body. The body fires a new pulse which travels out the axon to the entwined dendrite tips of other neurons. Though the dendrites have the function of receiving excitation from other cells, the cell-body also receives excitation directly, thus by-passing the dendrites. The synapse is the point at which an axon makes contact with the dendrite or cell-body of another neuron [Hebb, 1972].

The brain and nervous system are composed of millions of interwoven and intricately connected neurons. For some time, it was believed that the actual firing of pulses by the millions of neurons in the brain was the source material for what we know as an electroencephalogram (EEG). Today, the more popular belief is that the electrodes sense the general flow of current passing through the dendrites [Hebb, 1972].

The brain itself consists of primarily three parts: the brain stem, the cerebellum and the cerebral cortex. The brain stem and cerebellum are "white matter" areas while the cerebral cortex is mainly composed of "gray matter". This terminology was derived from the grayness of the neuron bodies and the whiteness of their axons. The spinal cord, brain-stem, and cerebellum are composed of neurons that have comparatively large and long axons. White matter areas are

primarily relay facilities, some transferring sensory information to the cerebral cortex and others carrying motor information from the cortex.

The cerebral cortex consists of two physically separated halves, called hemispheres. The two cerebral hemispheres are connected at their base by white matter. The cerebral hemispheres are essential to mental processes; their development constitutes the difference between lower and higher animals, and when they are removed nothing remains that can be considered thought or consciousness [Hebb, 1972].

Except for smell, each sensory surface (skin, retina, etc.) on one side of the body is directly connected with a cortical sensory area, specialized for that sense, on the opposite side of the brain. Although there are some connections with the same-side hemisphere, in general the paths are such that a stimulus event on one side of the body has its main effect on the opposite side of the brain. There are also two motor areas, one on each side of the brain, and these too have crossed over connections so that the right side of the cortex controls the left side of the body and vice versa. The remainder of the cortex, all that is not included in the specialized sensory and motor areas, is known as the association cortex.

Both cerebral hemispheres are subdivided into four major areas, called "lobes". They are: the frontal, parietal, temporal and occipital lobes. The division is on the

basis of physical configuration and function, but primarily the latter. Some of the dividing lines do not mean much psychologically or physiologically. However, two dividing lines are important: the sylvian fissure and the central fissure. The sylvian fissure is the deep cleft that partly separates the temporal lobe from the rest of the brain. The auditory area lies on the lower lip of the fissure. Also, man's speech area can roughly be described as the cortical region surrounding the sylvian fissure on one side of the brain — usually the left side. The central fissure is the dividing line between the frontal and parietal lobes; it also separates the motor area, in front of the fissure, from the somatosensory area, behind the fissure.

The somesthetic or somatosensory area on one side receives sensory messages from all parts of the body on the opposite side (plus some from the same side, but these are fewer and less important). Somesthesia means "body sensitivity", and includes sensations of touch, warmth, cold and itch from the skin; sensations of deep pressure and muscle tension and joint pressure, inside the skin; and sensations from the visceral organs [Hebb, 1972].

Scientists associate specific functions to the different lobes of the brain. Alpha waves are most likely to occur in the occipital lobe, which is linked to visual impressions, abstract thinking and conceptualization. The frontal lobe is considered the reflective contemplative area. Damage to the frontal lobe yields a different result nearly every time..

Often there is a lack of motivation, and some primary qualitative dualities may be lost. For example, the frontal lobotomy has been used to relieve intractible pain; after the operation, there may still be intense pain, but the patients are very apathetic about it. The temporal lobe is the center of sound and word formation. Damage in the temporal region often leads to hallucinations, loss of speech facilities, and, if the damage is deep within the lobe, loss of memory or confusion. The parietal lobes as well as having the function of primary body sense, appear to act as an auxilliary visual interpretive area, perhaps in correlating vision and touch. The results of parietal damage are less well defined.

Basically, the neurons of the brain act as on-off switches relaying pulses of current. The apparent variation in the frequency of brainwaves appears to be related to the synchronicity of the firing of these pulses. That is, as the pulses become more synchronous with each other, the EEG registers a lower frequency and a higher amplitude. The pulses in a particular area of the brain may be synchronous, while those of another area non-synchronous. Alpha brain waves are associated with synchronous pulses, while Beta brain waves are associated with non-synchronous pulses.

Brainwaves are classified according to four fundamental frequency ranges, and denoted by the Greek letters 'beta', 'alpha', 'theta', and 'delta'. Beta waves have frequencies

of more than 13 Hertz (HZ). The alpha range is generally considered to be from 8 to 13 HZ, theta from 4 to 8 HZ and delta less than 4 HZ. These four basic frequency ranges, although in common use, are broad categories that include a great variety of complex wave forms.

Beta waves are associated with mental concentration, anxiety, certain kinds of problem solving, attention, orienting and the jangled state most people feel from coping with the concerns of the everyday world. Alpha brain waves are generally associated with a relaxed, yet alert, mental state. Alpha brain waves are often predominant in the EEG's of Zen monks, Yogis and other Eastern meditators during the meditative state. Theta brain waves seem to be related to drowsiness, creativity and the dream portions of the sleep cycle. Delta brain waves are most prominent in deep sleep when no dream activity is present.

3. Alpha Research

In 1874, Caton discovered that the brains of monkeys and rabbits generated electric currents and that these currents varied when the subject was exposed to different external stimuli.

Hans Berger in 1929 used scalp electrodes to record EEG's in man. He found that the electrical currents varied both in amplitude and frequency. He attempted to relate certain brain patterns with mental states and suggested that the variations were related to changes in consciousness, such

as arousal from sleep. He is generally credited with discovering the alpha and beta rhythms.

The research in this area was greatly hindered for a time by the industrial revolution. Brain wave research lagged while the great thrust of science in this period dealt with "practical investigations", like atomic research. A further contributing factor to the lag in knowledge within this field was the primitive nature of the experimental instruments. For years scientists had to work with crude instruments as they studied the very subtle workings of the brain. With the advent of technological developments, new instruments were available and imaginative scientists were able to further research the frontiers of man's mind using biofeedback techniques.

Kamiya [1962] was one of the first investigators to attempt the study of operant control of EEG alpha and associated changes in mental activities. He was first interested in the question of whether human subjects could be trained to discriminate the presence or absence of alpha. His subjects were told that from time to time they would hear a bell ring; when they heard the bell, they were to make a guess as to whether, at that time, they were producing or not producing alpha activity. As soon as the subjects made their response they were informed if they were correct. The results indicated that by the third hour of training most subjects were correct 75 to 85% of the time. Some subjects became 100% correct in being able to name their brain wave state. Of

further interest was what the subjects reported about how they accomplished this task. "Any particular subject, even one who got 100% correct, was not necessarily able to articulate in English just how he was able to do this" [Kamiya, 1969]. This seems to suggest that Kamiya succeeded in teaching individuals to make internal discriminations or perceptions about their brain state whose dimensions were so unfamiliar that they were unable to give a clear-cut, verbal explanation.

Kamiya [1969] then directed his research to the question of whether individuals could be trained to control their alpha activity on command. He constructed an electronic device which would turn on a sinewave tone in the subject's room whenever the alpha rhythm was present. The tone would terminate as soon as the alpha rhythm disappeared. This alpha feedback methodology is called "binary feedback."

Kamiya began by training people to suppress the alpha rhythm without any training for enhancing it. Six of his seven subjects became quite proficient at performing this task. This experiment was followed by a study in both alpha suppression and generation. The results of this study showed a very significant difference in percent alpha time between the enhancement task and the suppression task; indicating some measure of volitional control over the alpha rhythm had been achieved by his subjects.

Dewar [1966] showed that people can be taught to voluntarily control their own alpha rhythms and use their EEG to send messages in Morse code.

Brown [1974] employed a blue light as a binary feedback signal instead of a tone. A blue light shone every time her subjects managed to produce alpha activity. The intensity of the light reflected the amplitude of the alpha. By the end of the first practice session, the average subject had more than doubled the amount of alpha in his EEG; he tripled the amount during the third practice session. She also showed that after giving subjects alpha feedback training, they could recognize and control their own alpha activity without any feedback signal.

Mulholland [1967] hypothesized that alpha activity was directly related to eye position. He believed that alpha activity increased when the eyes were moved to an extreme side or up position. However, Fenwick [1966] found no significant correlation between alpha and eye position as hypothesized by Mulholland. Additionally, Kamiya [1967] showed that his subjects could learn to control alpha with their eyes up or down. Thus, it would appear that the Mulholland effect cannot be generalized to all people.

Hord and Barber [1971] showed that volunteer subjects could learn in two 40-minute sessions to voluntarily increase or decrease their alpha activity with eyes open, using a contingent feedback tone as a guide. They also found that following their initial alpha feedback training, the subjects could perform "alpha control" tasks without any feedback.

Orne [1972] suggested that alpha feedback training could only help subjects to overcome influences in the external or internal environment, which were responsible for decreasing alpha density below the individual's inherent optimal level. Thus, the effect of alpha feedback training was likely to be demonstrated only under conditions where a suboptimal initial level was obtained. According to Orne, alpha feedback training taught an individual to increase his alpha density only under circumstances which normally caused it to decrease and, therefore, represented a kind of disinhibition. He believed that a variety of mechanisms could be responsible for this "alpha blocking". Thus, the alpha feedback phenomenon could be understood by recognizing that alpha augmentation depended upon learning to ignore or disregard the particular mechanism, which was responsible for decreasing alpha activity in a particular situation. Some of Orne's blocking mechanisms were severe stress, ambient light, sleep loss, pain, fatigue, and complex cognitive functioning.

However, Kamiya [1972] did not completely support Orne's inhibitory hypothesis and presented data which indicated that trained self-regulation of EEG alpha did result in the subjects' learning a new skill. Beatty [1972] also found that subjects learned to control occipital alpha and beta activity with equal ease in the dark and in the light, which he offered as contradictory data to Orne's hypothesis.

Although the effective control of alpha has been repeatedly demonstrated by subjects in different laboratories, one major question that is continuously advanced is whether the changes in alpha are due to progressive relaxation and acclimatization to the experimental situation. That is, an initial baseline measurement on subjects could have been biased due to the anxiety provoking nature of the experimental condition and not necessarily indicate a true alpha basal level. Therefore, subsequent increases in the alpha level over the initial basal level may not be due to any training, but merely due to the subjects being more relaxed and familiar with the experimental setting, thus producing more alpha.

Hart [1968] reported that a control group not given reinforcement showed increases in alpha, as did a contingently reinforced experimental group. Cleeland et. al. [1971] used yoked controls to examine alpha conditioning and reported no significant differences in amount of alpha generated by contingently reinforced and yoked control subjects at the end of training.

However, Kamiya [1972] noted that over four or five training sessions the post-session baseline alpha scores were lower than those during the tone feedback scores. This decrease in alpha without reinforcement indicated that even though there was an increase in baseline alpha over training days, this baseline increase was not as large as that observed during the actual feedback sessions. The higher alpha density

during the feedback session when compared to the baseline for that session denoted that the alpha enhancement was due to learning of some sort, but he was not exactly sure what was learned.

Travis et. al. [1974] argued that many of the past studies, which involved operant conditioning of alpha, did not adequately control variables which may lead to alternative explanations for the increase in alpha output. The two variables they referred to were: (1) alpha feedback signals that were artifactually elicited; and (2) spontaneous changes in alpha that were due to progressive relaxation and acclimatization to the experimental situation.

In one study measuring alpha under eyes-open conditions, Travis et. al. [1974], used three groups: (1) an experimental (contingent reinforcement) group, (2) a yoked (non-contingent reinforcement) group, and (3) a no-reinforcement group. The subjects were warned that some muscle tension and movement could turn on the feedback signal and were asked to refrain from such activities. They found that the subjects who received contingent feedback produced significantly more alpha than did yoked and non-reinforced controls.

Their results provide further evidence in support of the claim that a contingent positive feedback stimulus leads to increased output of alpha activity, and that the increases in alpha cannot be attributed to spontaneous changes or to artifactual effects [Travis et. al. 1972].

C. PAIN

1. Definition

We are all familiar with the concept of pain. Most of us dislike the experience which can be very intense. Pain is highly personal with each individual differing in his tolerance and conception. It is an abstract multidimensional concept which refers to (1) a personal private sensation of hurt; (2) a harmful stimulus which signals current or impending tissue damage; (3) a pattern of responses which operate to protect the organism from harm [Sternbach, 1968].

Pain is so familiar that we often take it for granted, but its acceptance does not lessen its importance. It is one of the most important symptoms encountered in medical practice and its reduction is a primary task of the physician. Despite the importance pain plays in everyone's life and the great interest it enjoys, it is surprising how little concrete knowledge actually exists regarding pain.

2. Historical Background

Originally, pain was regarded as an "unpleasant quality" associated with the sense of touch. It was a sign of something to be avoided, rejected or escaped from. Thus the origin of the viewpoint that pain was to be thought of as a warning or a signal of harm. Yet by no means is everything that is unpleasant, that is, disliked or rejected accompanied by the sensation of pain. Moreover, some sensory pains are actually pleasant and seem desirable; such as the pains that are welcomed by the masochist [Hardy, et. al. 1952].

In 1846, Ernst Weber ruled pain out of the sense of touch. He separated the sense of touch into the pressure sense, the temperature sense and the sense of locality. Pain, he placed with common sensibility, a catch-all category for those many vague organic perceptions that are known mostly by their biological functions - hunger, thirst, dizziness, nausea, and their like. For Weber, pressure, warmth, and cold are true sensations because they have their proper stimuli. Pain, on the other hand, seemed to have no proper stimulus but to represent a bodily need, like hunger or nausea.

In 1840, Johannes Müller presented his theory of specific nerve energies. He proposed five kinds of sensory nerves corresponding to the five traditional senses. The several types of nerves were thought to each carry a particular form of "energy" to the brain. Müller attributed a higher degree of specificity to peripheral nerves than would be acceptable today. The nerve pathways were considered to stand between the seat of consciousness and the external world. Müller identified the sensation with a specific neural apparatus, that is, for a sensation to be classed as such, it must be shown to have a functionally distinct set of afferent pathways and its specific integrative equipment.

In 1851, von Helmholtz succeeded in measuring the velocity with which the nerve impulse travels. This discovery

effectively put an end to one of the aspects of Müller's idea of "specific nerve energies" as it became apparent that all nerve fibers carry electrical impulses which differ only in magnitude, frequency and velocity [Hardy et. al. 1952].

Stimulated by Müller's theory, Blix, in 1884, discovered the sensitive points in the skin. Blix showed that the skin is not uniformly sensitive throughout. He further identified separate pain and pressure spots [Hardy et. al. 1952].

Von Frey not only studied the particular sensitive points but he also excised the skin beneath such points and by histological examination identified the specific end-organ type responsible for each sensation. Pain was conceived as being mediated by nerve fibers terminating in fine fibrils; cold by special large bulbous endings; warmth by the tightly coiled endings; and tactile sensation by networks of fibrillae contiguous with the hair follicles [Hardy et. al. 1952].

Although von Frey's formulation remains in many textbooks, other investigators were able to confirm the above observations only in the skin of the nipple and prepuce, but not in the skin over the forearm or other areas. This discrediting of the concept of end-organ specificity, reopened the question of whether or not pain has its special neural apparatus and if pain, according to Müller's doctrine could at all be considered a sensation.

Hardy et. al. [1952] observed that while such investigations were being carried out, three concepts of the

nature of pain stood in mutual opposition. The first one was called the "intensive theory" which was based upon the concept that pain was the result of intense stimulation of any sensory equipment. In support of this idea, Wundt assumed that the peripheral nerves of touch, heat, and cold were the only afferent pathways from the skin, as he saw no reason for assuming a special set of pathways for pain, or for considering pain a cutaneous sense. In his opinion the impulses from tactile or thermal stimulation, when reaching the spinal cord, found two pathways open: a primary low resistance pathway leading through the white matter, and a secondary high resistance pathway leading through the gray matter. Impulses of moderate intensity would take the primary pathway. If excessive impulses came, they overflowed into the secondary paths and passed upward to give rise to pain. His concept recognized the separate nature of pain sensation, but relocated the true pain fiber endings from the skin into the spinal cord.

The second concept held to the older emotion theory which supposed pain to be a phase of unpleasantness, an emotional state initiated by some sensation. These advocates viewed the neurologists as wasting valuable labor in the search for "pain paths" and for "pain localization" in the cortex of the brain, the paths in the spinal cord, and the supposed nerve terminals which have attracted the attention of investigators. For them, pleasure and pain were

two states which were too dissimilar to be commonly known by any one word, but so inseparable that they must be mentioned in one breath.

The last group supported the concept that pain was a sensation with its own distinct central and peripheral sensory mechanisms. Von Frey was considered the leader of this group whose views were adhered to generally by physiologists and physicians.

Nafe [1934] gave support to the intensive theory when he called attention to a possible relationship between the state of contraction induced in smooth muscles by varying the temperature of the stimulus, and the resultant sensations which von Frey had shown were experienced at these temperatures. Nafe implied that the sensation which was evoked was dependent upon the degree of contraction of the smooth muscle, which leads to a stimulation of the adjacent nerve endings — the more vigorous the contraction, the more intense the stimulation. The result was an alteration of the quality of the sensation from warmth, to heat, to pain; but all three being mediated by one and the same peripheral neural equipment. He inferred that pain was associated with the most intense stimulation. To Nafe, pain was the result of a summation of effects originating in intense smooth muscle contractions and integrated at the thalamocortical level. Furthermore, he implied that pain was similar to emotion and not a strict sensation, thus combining the intensive theory with the emotion theory.

Hardy, Woolf, and Goodell [1952] recognized the evidence supporting the old view that the feeling state may be the most relevant aspect of pain to the one who suffers. Yet, they emphasized that pain was a specific sensation with its own structural, functional and perceptual properties. They suggested that these two concepts do not oppose each other: "both represent attempts to formulate distinct but fundamental aspects of the pain experience." They proposed a fourth theory of pain to take into account the complex interaction of the components of the pain experience as well as the counterparts themselves. Their concept held the pain experience to be composed not only of pain sensation, but of associated sensations and of emotional and affective states as well. "Every sensation of pain (ache, prick, burn) is thus viewed as accompanied by a more or less predictable pattern of associated sensations (such as heat, cold, pressure) and feeling states (i.e., anger, fear, pleasantness, unpleasantness), the sensory resultant being the total pain experience for a particular individual." However, they viewed the pain sensation as the most important aspect of the total pain experience and associated phenomena were given secondary consideration in their studies. They advocated that there is a mathematically specifiable relationship between physical-stimulus intensity and pain intensity. Such psychophysical evidence was presented in support of the assumption that pain is a primary sensation arising from a physiological system that directly interconnects skin receptors and the pain center.

They held that the free nerve endings in the skin are considered to be the pain receptors.

A study by Melzack and Wall [1965] indicated the inadequacies of such a "specificity theory of pain" and presented an opposing theory of "patterning" (i.e. that pain is determined by stimulus intensity, input patterning, and central summation rather than by physiological specialization). This interpretation of the evidence led to their conclusion that the amount and quality of perceived pain are determined by many psychological variables as well as by sensory input. Observations by Beecher [1959] indicated that "activities in the central nervous system may intervene between stimulus and sensation which may invalidate any simple 'psychophysical law'." Beecher noted that during the battle at the Anzio beechhead in World War II, many severely wounded American soldiers entirely denied pain or perceived so little that they did not require medication for relief. Melzack and Wall explained this phenomenon by saying simply that these men felt no pain after their extensive injuries because the input was blocked or modulated by cognitive activities before it could evoke the motivational - affective processes that are an integral part of the total pain experience. They believed the assumption that pain as a primary sensation has relegated motivational and cognitive processes to the role of reactions to pain and has made them only secondary considerations in the whole pain process.

The traditional view of the pain mechanism failed to account for the fact that pain represented the result of at least two neuropsychological processes: (1) a sensory - discriminative process whereby stimuli are localized in space, time and along an intensity continuum, and (2) a motivational - affective component which drives the organism into activity aimed at stopping the pain as quickly as possible [Casey and Melzack, 1967].

Melzack and Wall [1965] further argued that the existing theories of pain could not account for the finding that the threshold for pain in response to shock on one arm could be raised by giving a second shock to the other arm [Halliday and Mingay, 1961].

The motivational - affective dimension of pain could be brought into focus when one considered the clinical studies on frontal lobotomies. Patients with frontal lobe lesions rarely complained about severe clinical pain and rarely asked for medication. Since a lobotomy did not disrupt sensory pathways, its predominant effect appeared to be on the motivational affective dimension of the whole pain experience. Both the aversive quality of the pain and the drive to seek relief appeared to be diminished [Casey and Melzack, 1967].

Melzack and Wall [1965] proposed a "gate control" theory of pain, which integrated the valid aspects of physiological specialization and patterning theory with what is

now known about central control of afferent input and spinal mechanisms. The gate control theory of pain provided the basis for considering the motivational dimension of pain in addition to its more obvious sensory dimension. They proposed that when pain occurs, selective brain processes were activated that exert control over sensory input via a central control trigger. The theory suggested that a gate control system in the spinal cord modulated the amount of input transmitted from the peripheral fibers to dorsal-horn transmission (T) cells which projected to the ascending fibers in the anterolateral cord. The number of impulses transmitted per unit time by the T cells was determined by the ratio of large and small fiber inputs, and by brain activities which influence the gate control system through central-control efferent fibers. The output of the T cells was monitored centrally over a prolonged period of time; when their afferent impulses reached or exceeded a critical intensity, the impulses triggered an Action System — those neural areas responsible for the complex, sequential patterns of behavior and experience characteristic of pain [Casey and Melzack, 1967].

This gate control system made it possible for central nervous system activities — subserving attention, emotion, and memories of prior experience — to alter afferent input. Their model intimated that psychological factors such as past experience, attention, and emotion influenced pain response

and perception by acting on the gate control system. The degree of central control seemed to be determined, in part by the temporal spatial properties of the input patterns. That is, some of the most unbearable pains, such as cardiac pain, rose so rapidly that the patient was unable to achieve any control over them. Conversely, more slowly rising temporal patterns were susceptible to central control and allowed the patient to "think of something else" or use other stratagems to keep pain under control [Melzack and Wall, 1965].

In addressing the question of whether pain is a sensory modality, Hilgard [1969] pointed out that if you cut your finger or stub your toe, pain did behave very much as if it were an ordinary sensory modality. That is, there was a stimulus, there were receptors in the fingers and toes, there was an afferent transmission of impulses, a central processing of the inputs, a perceptual response appropriate to the stimulus and a reaction to the stimulus. But yet, there were other considerations which made it less easy to assign pain the status of a sensory modality. Unlike pain, most sensory modalities had definite stimuli, definite receptors, specific sensory tracts, and localized receptive areas within the cortex. However, any stimulus could qualify to produce pain if it was intense enough; loud sounds and very bright lights are painful. According to Hilgard, the receptors for pain were unspecified, despite the role traditionally assigned to free nerve endings; and there was no one pain center localized

in the brain. Hilgard, at best, can only give us a qualified answer to the question whether or not pain can be counted as a sensory modality.

From a review of the literature on pain it becomes apparent that there are many and conflicting ideas on how pain should be defined and what comprises the "pain experience". Although the controversies have not yet ended, perhaps the best solution to the problem must be based on the combined evidence obtained by all the critical inquiries into the nature of pain. For, if it is at all possible, only in this way will the entire complex referred to as the "pain experience" be adequately explained.

3. Reactions To Pain

Paralleling the question of what comprises the pain experience is the question of individual differences in the reaction to the same pain stimulus. How do we account for the great individual differences in felt pain? Our interest in the realm does not overly concern the extreme cases of people who are born with practically a complete lack of sensitivity to cutaneous or other pains. Hilgard [1969] drew a comparison between this group of people to those born totally blind or totally deaf. Our interest lies within the normal population, wherein there are widespread differences in felt pain.

For instance, in the relief of postsurgical pain through morphine, Beecher [1959] found: (1) about a third of

the patients gained relief of pain through morphine that was greater than the relief following a placebo, (2) about a third got as much relief from a placebo as they did from morphine, (3) the final third were relieved neither by the placebo nor by morphine in doses considered safe to use.

Man is basically a social creature with a long period of development and dependence. He is dependent for his existence upon the aid, support, and encouragement of other men. Thus man's culture becomes the conditioning influence in the formation of his individual reaction patterns to pain. Furthermore, differences in pain responsiveness have been found to be associated with social class, ethnic groups, and family structure. For example, Gonda [1962] found that those from the working class complain more to the nurses in hospitals than do those from white-collar classes.

Pain responses in the laboratory appear to follow some of the theories of cognitive consistency, in that the pain corresponds to the amount of reward offered for participating in the experiment — the greater the reward the greater the pain — as though some suffering is consistent with the higher pay for participation [Zimbardo et. al. 1969].

In addition to the culturally conditioned reactions to pain, the meaning of pain to the individual may be affected by such factors as "habituation", "hypnosis", or "suggestion."

With respect to habituation, Hardy et. a. [1952] studied human responses to a thermal radiation pain stimulus and the associated pattern of change in the galvanic skin

response. They found that, although the initial response was extensive, during the first few months of the experiment the response to the painful stimuli became of lesser and lesser magnitude. Finally, the galvanic skin response to the same intense stimulus disappeared entirely. This process has been called "negative adaptation" or "habituation."

The observations on habituation were explained by Hardy et. al. [1952] in considering the reaction to the pains as indicating the degree of threat evoked in the subject by the pain. Initially, the entire procedure contained for the subjects an element of danger. As the experiments were repeated the threat content of the stimuli diminished.

The importance of habituation in the broad sense is obvious in that it provides a necessary flexibility to the organism in its struggle for survival. Otherwise, the individual could become overwhelmed by the reactions to symbols of danger, if coping/relief could not be achieved through habituation. Conversely, too much adaptation to pain or forms of threat might be disastrous. That is, while adaptation to painful or threatening stimuli serves to protect the individual by augmenting his flexibility, it may actually endanger him by making him sluggish under conditions where he should focus on the pain and its causes, vice ignoring it.

Hypnosis is an intermediate state between waking and sleeping; it is partial sleep, a partial inhibition. Kubie

and Margolin [1944] described the hypnotic state as one in which the subject's ego boundaries, previously constructed, are partially expanded and incorporate an image of the hypnotist. This image, which echoes the hypnotist's voice, is a part of the subject's new and temporary ego, and thus hypnosis reproduces the developmental process in which the child incorporates an image of the parental figure. In the hypnotic state, because of the incorporative process, the subject's behavior and subjective experiences seem to come from himself, rather than from the hypnotist.

Gill and Brenman [1959] offered a similar view, but stressed that two kinds of regression are involved; one is an altered state of consciousness, in which the subject loses some of his autonomy and part of his ego is dominated by the social environment; the other is that the subject engages in a regressive relationship with the hypnotist. Thus, hypnosis is both an altered state and, a transference relationship, and the two phenomena are complexly interrelated.

The first extensive use of hypnotic analgesia in surgery was reported by Esdaile in 1846 [see Boring, 1957]. Since then the literature in medicine, surgery, and hypnosis has been filled with many reports on the successful use of the technique in many different kinds of procedures. Among the well-documented effects of hypnotically induced analgesia are: the absence of all signs of pain; diminished bleeding; diminution or absence of postoperative shock reactions; greater speed of recovery, etc.

One major question that still remains to be answered is whether it is the hypnotic state per se that causes the diminution of perceived pain or whether it is caused by the 'suggestions' of pain relief given in a close interpersonal setting.

Assuming that the galvanic skin response provides a relatively accurate measurement of the autonomic response to the threat of pain, Hardy et. al. [1952] found no evidence to support the hypothesis that the hypnotic state per se affects this response. However, their own experimental results left no doubt that hypnotic state coupled with the suggestion of anesthesia diminished the galvanic skin response to pain.

Barber [1962] went so far as to suggest that "hypnotic analgesia" at times produced not a reduction in pain but an unwillingness to state directly to the hypnotist that pain was experienced or a temporary "amnesia" for the pain was experienced. There is a strong motivation for the denial of pain present in the hypnotic situation. The physician has invested time and energy hypnotizing the patient and suggesting that pain will be relieved. He further expects and desires that his efforts will be successful and communicates his desires to the patient. The physician has structured the situation such that even though the patient may have suffered, it is at times difficult or disturbing for him to state directly to the physician that pain was actually experienced.

Barber suggested that caution is necessary in accepting the hypnotic patient's verbal report or lack of overt behavioral reactions as valid indices that the patient did not suffer. He contended that an objective index of pain was needed in studies concerned with hypnotically suggested analgesia. Barber's objective index would consist of one or more of the following: blood pressure, heart rate, respiration, digital vasomotor tone, skin resistance and degree of tension in localized muscles. A series of experiments that monitored the physiological responses which are normally associated with painful stimulation found that in some instances "hypnotically suggested analgesia" reduced some physiological responses to noxious stimuli and in other instances physiological responses were not affected. In one experiment physiological reactions to painful stimulation were compared under (1) "hypnotically suggested analgesia" and (2) a waking condition in which subjects were instructed to imagine a pleasant situation when noxious stimulation was applied. It was found that both conditions were equally effective in reducing subjective and physiological responses to painful stimulation [Barber and Hahn, 1962].

Barber's review suggested that the two critical variables in hypnotic analgesia were actually; (1) suggestions of pain relief and (2) the close interpersonal setting in which they are given. He emphasized that further experiments in the area of effects of hypnosis on pain should control:

(1) the preexisting level of suggestibility among subjects assigned to the 'trance' and control treatments; (2) the interpersonal relationship between subject and experimenter; (3) the suggestions of pain relief per se [Barber, 1971].

In opposition to Barber's steadfast adherence to physiological indicators serving as the most accurate indices of pain, Hilgard [1969], questioned whether there presently existed any satisfactory physiological indicators of pain. For Hilgard, a satisfactory physiological indicator of pain was one which was present (or increased) when pain was felt, and absent (or reduced) when pain was not felt. Furthermore, the correlation between the physiological indicator and the verbal report had to be established both positively and negatively if the indicator was to be used in confidence in the absence of a supplementary verbal report. He summarized the state of our knowledge of pain by simply saying that, "at present, there was no single accepted indicator of pain that can be counted to vary in an orderly way with degrees of pain."

In his experiments on pain, Hilgard [1969] used two sources of noxious stimulation. In the first one, pain was produced by placing the subjects hand and forearm in circulating cold water at several temperatures. This procedure is commonly referred to as the cold pressor test [Wolf and Hardy, 1941]. In the second method, pain was produced by first placing a tourniquet just above the elbow, and then

instructing the subject to squeeze a dynamometer a standard number of times. After he stops exercising, the pain begins to mount and eventually the forearm becomes quite painful. This is called ischemic pain [Smith, et. al., 1966].

While the hand the forearm were immersed or while the tourniquet was applied, the subject reported he felt pain on a scale of 0 to 10, 0 being no pain and 10 being the point at which he would wish to remove his hand or have the tourniquet removed: Such verbal pain reports were proven to yield an orderly relationship to the conditions of stimulation, in the sense that the pain reported bears a systematic relationship to the temperature of the water and to the time of exposure to the noxious stimulation.

Hilgard [1969] emphasized these findings as a reply to those who would degrade the subject's statements as being merely verbal reports, as though some sort of physiological response would be sounder. He flatly asserted that there was no physiological measure of pain which is either as discriminating of fine differences in stimulus conditions, as reliable upon repetition, or as lawfully related to changed conditions, as the subject's verbal report.

To support this assertion, Hilgard studied pain reduction under hypnosis for both the cold pressor test and ischemic pain using both verbal reports and systolic blood pressure as indices of felt pain. In the normal waking condition, the rise in pain as verbally reported in the cold water was

accompanied by a rise in blood pressure. Likewise, the rise in ischemic pain verbally reported was accompanied by a rise in blood pressure. Thus he established systolic blood pressure as a candidate to serve as an indicator of pain.

First, considering the reduction of cold pressure pain, it was found that hypnosis alone did not appreciably reduce the pain as verbally reported by subjects. However, hypnosis with suggested analgesia did indeed produce a reduction in verbally reported pain. The verbal pain reports thus yielded an orderly picture of pain reduction under hypnotic analgesia. With respect to the blood pressure measures, it was surprisingly found that the blood pressure still rose independent of the amount of felt pain. Hilgard [1969], concluded that when pain is felt in the cold pressor experiment, in the normal waking state, there is a tendency for blood pressure to rise in an amount correlated with the amount of experienced pain, but when combined with hypnotic analgesic suggestions blood pressure may rise in a stressful situation independent of the amount of pain actually experienced. From this finding, Hilgard asserted that blood pressure is not a completely satisfactory indicator of pain.

The relationship between systolic blood pressure and pain reduction with hypnotic suggestion turned out differently under ischemic pain. Under hypnotic suggestion subjects were able not only to rid themselves completely of pain for a matter of 18 to 45 minutes, but their blood pressure,

which rose sharply in the waking state, did not rise or rose very little [Hilgard, 1969].

Hilgard concluded that the absence of pain reported by the subject under conditions of hypnotic analgesia was sometimes confirmed by the absence of a rise in blood pressure. Thus, he had a physiological validation for the reality of hypnotic analgesia, but the validator was only in one direction. That is, absence of the blood pressure rise may be taken as an indication of the absence of pain under specified conditions, but pain may be absent even if blood pressure rises. It is for this reason that he asserted blood pressure and other physiological measures were not completely satisfactory indicators of pain and should not be used in lieu of the subject's verbal report but rather in conjunction with it.

Sternbach [1968], in discussing the data on hypnosis relevant to pain, stated that hypnotic induction typically involved the subject's being immobile and attending only to the hypnotist's instructions (constricting sensorimotor input). In the hypnotic trance the subject was convinced that his experiences were as the hypnotist said; it was as if the hypnotist became a part of the subject. For the subject to be able to accept these conditions, he must be willing to allow himself to be helpless and to trust the hypnotist. Consequently, the fact of being hypnotized already indicated that the subject was able, even if only temporarily, to (1) focus

his attention, and (2) give up feelings of anxiety about himself.

From such data it can reasonably be inferred that such hypnotic analgesia was effective either because attention was focused elsewhere (other directedness), or because anxiety (concern about the stimulus effects) was very low. Sternbach based this inference on the data provided from the control subjects of the experiments, for whom hypnotic analgesia was not used, but yet their responses to the noxious stimuli were as minimal as subjects in the trance condition.

However, Sternbach [1968] made a further inference concerning the relative roles of attention focusing and anxiety reduction. It was his impression that the focusing of attention was not in itself essential to the elimination of pain. It was necessary for the induction of hypnosis, and it was a useful means for a subject to gain control over anxiety concerning pain stimuli. However, the data strongly suggested that in hypnotic analgesia, as well as in other conditions, it was the absence of anxiety about the stimulation which was the single necessary and sufficient condition for perceiving the stimulus as a nonpainful sensation.

4. Anxiety And Hypertension

Pain is not a necessary prerequisite to anxiety. Anxiety can be defined in a number of ways. A "normal" level of anxiety is necessary to keep us alert and prepared to take action. Neurotic anxiety is a condition unrelated to

any specific situation. This is said to be the "trait" of anxiety as opposed to situational anxiety. Operating on either the conscious or unconscious levels, anxiety can be physiologically or psychologically induced.

Hans Selye's concept of systemic stress is that stress is not only the result of some external stimulus but is also affected by the somatic response to the stressor. Systemic stress is then manifested by the General Adaptive Syndrome (GAS) which is the body's response to a stressor through an alarm reaction. This reaction takes the form of a shock phase during which resistance is lowered, followed by a countershock phase which sees the activation of somatic defensive mechanisms. The body's resistance to the stressor is increased to the point where maximum adaptation is reached. If this adaptation is insufficient to counter the stressor, exhaustion will occur and the adaptive reaction collapses. The alarm reaction is characterized by excitation of the Autonomic Nervous System (ANS). The physiology of this reaction appears as adrenal discharge, blood content changes, heart rate and muscle tone changes, perspiration, arterial constriction and pooling of blood in muscles. The body is said to be aroused. On the mental level, the stimulus is perceived by the sensory receptors. Memory is searched to determine if the situation is physically or psychologically threatening through comparisons with similar situations. If the situation is not threatening or if the brain devises an

effective defense, the ANS response subsides and arousal decreases. If, on the other hand, the stimulus is perceived as endangering, the "fight or flight" response or a modification thereof appears and is accompanied by elevated anxiety. Situations in which the stimulus is perceived to be life-threatening may transcend anxiety to the point of blind panic.

If a stimulus presents an actual threat to the physical, mental or emotional self, the anxiety reaction is beneficial in that it alerts the body for action. If the situation is not threatening but the alarm reaction proceeds, an imbalance occurs and homeostasis needs to be restored. If this mediation does not occur, a pervasive anxiety results. This form of anxiety seems to be fairly prevalent in our society. On any given day, we are bombarded with news of bombings, riots, wars, economic crises and so forth. Through the years, we have become so acclimated to these common occurrences that in most cases the information registers largely on the subconscious level such that we are unaware of the resultant anxiety. Consequently, many Americans seek relief through drugs or alcohol.

So much of an individual's reaction to stress hinges on his past experience that, as a result, responses are highly varied. There appear to be several common physiological indicators of anxiety. Evidence seems to indicate that these responses assume some pattern. These measures are generally heart rate, galvanic skin response, blood pressure,

adrenal discharge, muscle tension, respiration rates and other chemical analyses.

It can be safely stated that except for sudden life-threatening situations, no stimulus is a stressor to all individuals. This conclusion is based on the findings of Miller [1953], Appley [1962], and Lazarus, Deese, and Osler [1952] among others. These variations as to what is a stressor to an individual are often due to our "social shaping" and our response will be similar whether based on factual information or not. Acrophobia, for example, is a very real fear to many individuals yet one would hardly expect such a fear in a skydiver.

A number of studies have produced some general observations of stress in relation to anxiety. As indicated, individuals vary in their response to a given stressor. Some quickly show signs of stress, others show improved performance as a result of increasing alertness while still others show no reaction. Many varied external conditions can cause stress. Knowledge of the stressor alone does not lead to prediction of the intensity of the stress reaction. Other factors such as motivation and history need to be taken into account.

Hypertension has received a great deal of attention in the last decade. Physicians refer to essential hypertension as elevated blood pressure with no specific cause. It is believed to be the result of being nervous and unable to relax and may also be a result of such things as kidney

disease, adrenal gland tumors, and pinching of the aorta (coarctation). Hypertension is directly connected to strokes and heart attacks and an estimated 60,000 Americans die annually, with hypertension listed as the sole cause of death. Hundreds of thousands of others will suffer strokes or heart attacks with hypertension as a major contributing factor.

Military personnel have always been excellent candidates for hypertension. Combat situations often produce fatigue or exhaustion, loss of sleep, pain and anxiety as a result of concern for self and others. This situation can lead to acute stress degrading the individual's performance and health. Anxiety adversely affects the ability to sleep leading to fatigue, more stress, and more anxiety.

Peacetime occupations of military managers also lead to stress. Project managers are responsible for hundreds of millions of taxpayer dollars. Commanding Officers of Naval vessels and squadrons having responsibilities measured both in lives and in dollars and these are but a few examples. Anxiety, held to a proper level, can enhance their performance. At higher levels, anxiety is counterproductive and can cause a decrement in performance, and quite possibly result in early death.

Probably the most disheartening fact about hypertension is that there is usually no warning of its presence and only a blood pressure check or actual dysfunction involving the heart, eyes, brain or kidneys reveals its existence.

Treatment initially involved unappealing salt free diets. Today, drugs are used extensively in the treatment of hypertension. Pentaquine, chlorisondamine, reserpine, and others are used for symptomatic relief of hypertension. However, some drugs used in the treatment can cause, along with lowered blood pressure; impotence, dizziness, and drowsiness. A further problem is that the effects of these drugs are beyond the control of the patient. This is to say that the drug could cause drowsiness when a situation confronting the patient calls for alertness. The body is prevented from responding in an appropriate manner. A more optimal approach would be to give the patient the ability to deal internally with his own level of stress.

Miller and Di Cara [1970] have performed a number of experiments indicating that standard stress response such as increased heart rate also increase anxiety and the effects of stress. The ability to control these responses would seem to mediate the effects of stress. The relaxed subject when confronted with a stressor, usually exhibits certain physiological responses such as increased heart rate and blood pressure, increased palmar skin conductance, constriction of the blood vessels and a large reduction in Alpha brainwave activity. It would appear logical, then, that a person moving from an anxious state to a relaxed state would exhibit the opposite physiological reaction.

A method of relaxation that can be turned on and off at will would seem ideal. Jacobson devised a technique

in 1938 of progressively relaxing the muscles of the body. A similar method has been used to combat anxiety [Haugen, Dixon & Dickel, 1963]. The problem encountered by these methods is the relatively large amount of time required to learn them (up to 2 years). Biofeedback, with its proven accelerated learning potential, was used by Budzynski, Stoyva and Adler [1970] in experiments designed to cure tension headaches. They utilized electromyograph (EMG) feedback. Electrodes attached to the frontalis muscle in the forehead relayed, through biofeedback machinery, the level of tension in that muscle. This constant reading provided sufficient information for the subject to relax that muscle. They found that other muscles such as in the neck and shoulders also relaxed. Their tension subjects showed a dramatic decrease in the intensity and frequency of headaches. Other researchers and clinicians have utilized several forms of biofeedback concurrently to achieve deep relaxation such as EMG, EEG, and body temperature feedback [Green, E.E., et. al. 1969]. Green and his associates were able to reduce muscle tension to near zero levels, achieving deep relaxation with a very small amount of training. Once learned, the ability of the subjects to mentally require their bodies to relax was retained without the need for instrumental feedback. At the same time, unlike drug induced relaxation, mentally induced relaxation can be turned off if a situation demands attention.

As previously mentioned, a certain amount of anxiety is necessary to help us function in this society. Orne,

director of experimental psychology at the University of Pennsylvania, believes that each of us has a level of anxiety at which we function best. Either too little or too much anxiety degrades performance. Extreme relaxation attained through EMG feedback may place us at a level of anxiety too low for adequate performance at tasks. Kamiya's [1969] previous finding that muscle relaxation is not necessary to produce alpha brainwaves but that relaxation often occurs during the alpha experience and that most high alpha producers are relaxed individuals, suggests that alpha brainwave training is a viable approach in reducing anxiety.

5. Anxiety Component Of Pain

Anxiety is usually specified as the single most important component in the reaction to pain. Like pain, it must be treated as a construct in that its presence can only be determined by responses of the subject whether verbal and/or physiological.

One study [Schalling and Levander, 1964] compared sensitivity to pain from electric shock between a group of anxiety prone delinquents and a group showing predominantly psychopathic traits. The anxiety prone group was found to be more sensitive to pain. Damaser, Shor, and Orne [1963] used hypnotic analgesia to reduce anxiety and concluded that elimination of the anxiety component caused elimination of the normal physiological responses associated with the pain stimulus. Beecher [1955] reported that there is no dependable correlation between degree of pathological injury and the

degree of pain experienced. Malmo and Shagass [1949] found that "patients with anxiety seem to have a low threshold for sensation of pain, or at least they respond with withdrawal and with signs of motor disturbance to lower intensities of stimulation than do normal subjects". Thurlow [1962], through a comprehensive literature review, cites evidence that both "susceptibility of people to illness, and illness behavior such as the use of medical facilities, is related to personality traits and also to psychological stress." Bond and Morgenstern [1967] found that anxiety increased with chronic pain. Merskey [1973], in a study dealing with pain, found that "... it arises very frequently in conjunction with neurotic illness and is made worse in circumstances which promote emotional tension." Merskey further states that "the threshold (of pain) is also usually thought to vary somewhat with sex, occupation, cultural attitudes, ethnic group and mood. Thus, women tend to have lower thresholds than men, labourers and miners have higher thresholds than clerical workers and anxious patients have lower thresholds than those who are not anxious." A study conducted by Shannon, Szmyd, and Prigmore [1962] examining Adrenal Cortical Hydroxycorticosteroid (ACTH) responses in patients undergoing dental procedures, including surgery, showed that anticipation of the procedures produced an anxiety response. They stated, "Since the pituitary-adrenal system is generally stimulated under conditions in which the integrity of the organism is threatened, it might be expected that fear or anxiety most

likely would be associated with increased ACTH release." Adler and Lomazzi [1972] found support for the belief that pain is a perception determined by the individual's past history, the meaning of the stimulus to him, his "state of mind", and by the sensory nerve patterns evoked by the pain stimulus. Furthermore, studies have shown that the only necessary criteria to elicit maximum pain responses is that anxiety also be high. This anxiety need not be related to the intensity of the stimulus or even the degree of injury.

A large amount of inquiry into the relationship between pain and anxiety has resulted in a number of interesting findings. Lynn and Eysenck [1961], in examining the personalities of their subjects, predicted that extroverts will show less reaction to pain than neurotics due to a lower anxiety component. A contradictory study [S.B.G. Eysenck, 1961] using verbal assessment of the labor experience in 200 women indicated that extroverts tolerate pain better but that they tend to verbally exaggerate the experience. Studies conclude that anxious subjects show a lower tolerance to pain [Hare, 1965; Merskey, 1965]. Anxious behavior can be described physiologically, behaviorally or affectively and the individuals perception of pain results from his capacity for and method of dealing with anxiety. An additional problem enters when the method used enhances rather than mediates anxiety and the increased anxiety results in greater responses to pain.

We are thus left with a number of inferences as to how to deal with pain when normal medical or surgical procedures fail to bring relief. (1) In those cases where there is no stimulus or tissue damage, the subject's affective description will probably be one of depression. Alleviation, whether through electric shock, psychotherapy, or anti-depressant drugs usually brings pain relief. (2) When tissue damage or a stimulus is present, the affective description is usually one of anxiety, the reduction of which usually brings relief. (3) There are a number of methods utilized to reduce anxiety. These methods might involve altering the pattern of the stimulus, hypnosis, placebo intervention, or focusing attention through 'other-directedness' such as engaging in a task. All have the property of eliciting responses incompatible with painful or anxious states.

(4) The implications are that a viable method of alleviating pain is to interrupt the anxious response. There are two approaches to this intervention. The first is to lower the level of anxiety prior to the painful stimulus and the second is to reduce anxiety in the person already experiencing pain.

6. Placebo Phenomenon

Further evidence relating to approaches in the reduction of anxiety and its concomitant reduction of perceived pain can be derived from an analysis of the placebo phenomenon. One form of placebo may be a pharmacologically inert substance formerly used to "please" patients more than to help them,

now used extensively as a control in experiments which examine the effects of drugs. The placebo often produces relief of any or all symptoms for which it is administered, including relief of pain. Additionally, this inactive agent frequently produces side effects, some of them toxic in appearance. Moreover, the reaction to the placebo is not limited to alteration of the mental states but can also produce observable physiological responses [Sternbach, 1968].

Two themes occur repeatedly in studies of pain relief through hypnosis and placebos. One is that anxiety reduction and pain relief are associated; the other is that neither hypnosis nor placebos are particularly effective in producing pain relief if anxiety is not present in some minimal amount, or if it is kept high due to other factors [Sternbach, 1968].

While anxiety reduction may be one indication of the effectiveness of hypnosis and placebos, it seems likely that other psychological processes are operative. Some writers have conceptualized placebo administration as a social influence situation in which many factors are involved. "The placebo response may be viewed as a direct function of the 'stimulus'; however, the 'stimulus' is not the ineffective inert compound but the entire situation which includes the 'drugs', the words of the physician, and the patient's previous experience with physicians and drugs" [Barber, 1959].

7. The Classical Conditioning Paradigm

The experience of pain is often associated not only with accidental injury, but, in childhood, also with punishment. Therefore, there is an association among the emotions of pain, guilt, fear, etc. Sternbach [1968] points out that the relief of pain is typically associated with comfort, love, expressions of caring, and with the reduction of anxieties related to the presence of love. We observe this phenomenon when the 'hurt' of a child tends to subside upon receipt of the mother's affection.

The classical conditioning paradigm outlined by Hernstein [1962] suggests that whatever in the past is associated with pain relief will tend to acquire the property of inducing such relief. In man, there are several such associations, and the patient typically is exposed to more than one: the doctor, medicines, comforting behavior, etc. These all involve the patient in a passive, dependent, regressed relationship with others whether as a hypnotic subject or as the recipient of medication. For many patients, such a childlike role is sufficient to induce the reduction of anxiety and relief of pain, and these patients will also probably be the placebo reactors and good subjects for hypnotic analgesia.

8. Pain Reduction in Obstetrics

Presently, the most concentrated and successful attempt to incorporate all three effects of suggestion, focusing attention, and anxiety reduction into one program

of pain relief has been in the field of obstetrics. Throughout history man has sought to diminish or even to suppress, the pain of childbirth, at first by magical means, later by more scientific methods. In the nineteenth century, modern medicine introduced anaesthesia and analgesia produced by chemical substances. However, these pharmacological methods were not free from toxicity for both mother and child, and moreover suppressed an important emotional experience in the woman's life. Therefore, other methods were sought after. About the beginning of the nineteenth century, psychologically produced analgesic was demonstrated experimentally using hypnosis. Childbirth without pain was carried out under hypnosis, but only on a small scale. Hypnosis was shown to have a particular psychotherapeutic effect, but as Chertok [1959] indicates, it always aroused prejudice and does so still. Under these circumstances new methods were developed which did not directly rely on hypnosuggestive techniques. The first was that of Read, and the second was the work of the Russian psychiatrist, Velvovski [Chertok, 1959].

For Read, civilization and culture brought influences to bear upon the minds of women which have introduced real fears and anxieties concerning labor. The more cultured a particular race became, then the more dogmatic they have been in pronouncing childbirth to be a painful and dangerous ordeal. This fear and anxiety gives rise to natural protective tensions in the body, and such tensions are not of the

mind only, but includes the protective mechanism of muscular tension. Unfortunately, the natural tension produced by fear and anxiety influences those muscles which close the womb and prevent the child from being driven out during childbirth. Therefore, fear and anxiety inhibits the birth process; that is, it gives rise to resistance at the outlet of the womb when in the normal state those muscles should be relaxed and free from tension. Such resistance gives rise to real pain because the uterus is equipped with organs that record pain caused by excessive tension. Thus, the Read Method viewed anxiety, tension, and pain as external influences which were not normal to the natural design of childbirth but which were introduced in the course of civilization's development. If pain, fear, anxiety, and tension go hand and hand, then it is necessary to relieve the tension and to overcome the fear and anxiety in order to reduce or eliminate pain. The implementation of Read's theory is demonstrated in the methods by which anxiety and fear may be overcome, tension may be eliminated and replaced by physical and mental relaxation.

Applications of such methods as Read's and the psycho-prophylactic method of the Soviet Union are practiced today throughout Europe, South America, Africa and the United States, as well as many eastern countries under the title of the Lamaze Method.

Basically, the Lamaze method prepares a woman emotionally, intellectually, psychologically, and physically

for childbirth. The trained woman approaches childbirth with a positive attitude and accurate expectations. Accurate expectations about sensations of pain were shown by Johnson [1973] to reduce the incongruency between expected and experienced sensations and was also associated with less intense emotional response during experimental pain stimulation. Additionally, accurate knowledge of what was going to transpire had a strong influence on reducing anxiety, fear, and in turn, the tension which intensified the pain experience.

The principal physiotherapeutic method used in the Lamaze method is a progressive relaxation technique accompanied by breathing exercises designed to facilitate relaxation. For Read, body relaxation must be recognized as a necessary phenomenon and should be accompanied by a mental indifference to the uterine contraction. It is further believed by many American authors that relaxation exercises often produce hypnoid states which make the patient more susceptible to analgesic suggestions [Chertok, 1959].

The respiratory exercises are considered as processes which reduce pain by focusing attention on the respiratory mechanism. Furthermore, it is believed the increased oxygenation and respiratory rhythm itself assumes an important role in achieving self-analgesia. Chertok [1959] believes there is a clear relation between respiration and the emotional states which find their expression via the respiratory system. He emphasizes that respiratory factors are important in Yoga and the Yogis themselves are able to produce

complete anesthesia. Moreover, in examining the EEG's of women who underwent psychoprophylactic (or mind-prevention) preparation, it was found that their calm behavior during the delivery was also reflected in their brain wave patterns. The changes in the alpha rhythm during the contractions were minimal, indicating an ability to hold a relaxed state of mind throughout labor and delivery [Chertok, 1959].

Everything in the method is directed to the suppression of fear and anxiety. Read stresses as most essential in achieving this goal is the establishment of a good interpersonal relationship between the doctor and the patient. As to suggestions, they are considered by many to be one of the greatest and most harmless anesthetizing agents the obstetricians have. The very expression "painless childbirth" can have an extremely powerful suggestive effect on women.

9. Relaxation, Alpha Production, And Pain Reduction

From a review of the literature, it is apparent that the ability to reduce a subject's level of anxiety and hold a deep relaxed state offers tremendous aid in coping with a painful stimulus. Several studies indicate that the alpha state may aid in achieving this end. Saul et. al. [1937] found that a high alpha index (percent of time the subject was in the alpha state) was closely associated with "passivity". McAdam and Orme [1954] determined that subjects registering neurotic scores on the ranking Rorschach test tended to have a low alpha index. Ulett, Gleser, Winokur, and Lawler [1953] and Brockway, Gleser, Winokur and Ulett

[1954] found that anxiety is associated with a decreased alpha index. As previously cited by Shannon, Szmyd and Prigmore [1962], the release of ACTH is an indication of cortical arousal as a probable result of fear or anxiety. Moruzzi and Magoun [1949] found that the degree of "alpha blocking" is a measure of cortical arousal. The implication is that fear and/or anxiety are detrimental to the generation of alpha brain waves. Davidson and Neufeld [1973] using muscle tension and respiration rate as physiological measures found that "relaxation procedures are more effective than cognitive rehearsal procedures in increasing pain tolerance." Lindsley [1951] determined that patients with pathological anxiety showed "... a low level, fast frequency EEG pattern." This can be contrasted with the slow frequency of the alpha brain wave. Chertok [1959] emphasized that complete body and mental relaxation are a necessary concomitant to 'painless childbirth'. When generating alpha brain waves, most people express the feeling of relaxation. Kamiya [1969] reported that his subjects associated some kind of relaxation of the mental apparatus with the high alpha state. Brown [1974] reported that, in her experiments, subjects described the alpha experience as quite pleasant, a feeling of comfortable relaxation. Kamiya [1969] described the good alpha subject as a person who appears interested, relaxed, and comfortable. Although muscle relaxation was not proven to be directly related to the mental relaxation associated with alpha production, Kamiya [1969] found that muscle relaxation often flowed right along with the alpha experience.

Green [1969] and Stoyva and Budzinski [1972] found that extremely anxious people had difficulty producing alpha brain waves, and did not reach that alpha feeling of tranquility and relaxation.

The inability to produce alpha may be related to how anxious people look at life. Lawrence [1972] points out that this statement can be taken literally and is concerned with the visual functions of the anxious individual. This person usually has the characteristic of rapidly searching eyes ('looking behavior') which somehow acts to block the alpha brain wave.

It is the contention of many alpha researchers that, through biofeedback training, a normal person can quickly, efficiently and thoroughly learn how to relax; simultaneously reducing his anxiety to a level where he can better function [Lawrence, 1972].

Once a person is able to achieve high alpha activity, the lack of tension seems to follow as a matter of course. As previously indicated in Read's theory of 'natural child-birth', if anxiety can be reduced then tension can be overcome and pain will be reduced or eliminated from the child-birth experience. Fehmi [1969] points to the classic studies of both Yoga and Zen masters who demonstrated large-amplitude alpha brain waves and extremely low levels of muscle tension.

Additionally, Anand et. al. [1969] showed that during samadhi (meditation), two Yogis were able to keep their hands in water at 4° centigrade (cold pressor test) for 45-55

minutes without experiencing any discomfort. The EEG records of these two Yogis showed persistent alpha activity both before and during the period in which the hand was immersed in the water. Their ability to maintain alpha and not experience discomfort suggested that, while meditating, these individuals were somehow able to block the afferent impulses. However, when not meditating, they were unable to block these afferent impulses from external stimulation.

Of further interest was the fact that while the meditating yogis showed no response to external stimulation, the Zen monks did respond. Tart [1969] suggests that this difference may be due to the differing philosophical outlooks of Zen and Yoga. The Zen monks strive to exist in the here and now, "in the immediacy of the phenomenal world". Therefore, their response to external stimulation could be viewed as their successfully managing to stay in the here and now of immediate sensory experience. On the other hand Yoga philosophy has a strong "world-denying quality". The yogin strives to transcend the phenomenal world which is considered all illusion and ensnarement. Therefore, it would make sense that they showed no EEG response to stimulation and no recall of the stimulation after meditating.

At New York University, Torres demonstrated his no-pain abilities to EEG feedback researchers, using his own version of South American Yoga [Lawrence, 1972]. He stuck a sharpened bicycle spoke through his cheeks while showing no indication of pain. His EEG records indicated that while

he was performing this feat he was generating high amplitude alpha similar to yogi and Zen masters EEG's.

Kamiya [1969] demonstrated that ordinary subjects may be trained by biofeedback techniques to produce an EEG pattern similar to that found for the meditating Zen monks and Yogins, that is, almost continuous alpha. As Tart [1969] states, "while it would be naive to equate the state of consciousness of the meditating Zen monk or yogin with that of the college student producing almost continuous alpha rhythm, the fascinating possibility is suggested that one of the things that Zen monks and yogins learn to do in their years of meditation is to produce a high alpha state. If we can produce the high alpha state in just hours in a modern psychophysiological laboratory, would our subjects have a pronounced head start if they then attempted to learn the practice of meditation in the Zen or Yoga style?"

Going one step further, some authors believe that besides training normal subjects to produce alpha activity similar to Yogis, biofeedback may enable the normal person to duplicate the Yogis no pain feats. "These no-pain brain levels would allow someone to undergo serious surgery without the debilitating effects of anesthesia" [Lawrence, 1972]. For example, the relaxation required in natural childbirth may be enhanced from the addition of alpha control feedback training.

Recently, Melzack [1972] investigated the possibility of using self-regulation, particularly alpha feedback training,

as a method to provide an effective technique for the control of pain. He feels that at least four variables can contribute to pain relief in the alpha training procedure: (1) distraction of attention from the painful body site to a particular inter-feeling state and to a feedback signal during training, (2) strong suggestions that the procedure will effectively diminish pain, (3) the relaxation that accompanies the alpha state produces a general decrease in arousal inputs, as well as a decrease in anxiety, and (4) the development of a sense of control over pain is known to diminish pain. Melzack's initial strategy was to utilize all of these variables in combination, including relaxation, suggestion, hypnotic instruction, and alpha feedback.

For Melzack's study, two groups of subjects were used; (1) clinical patients suffering from chronic pain, and (2) student volunteers. The clinical patients had suffered chronic back pain for several years and their pain had persisted despite surgery, psychiatric counseling, or one or more of the standard physiotherapeutic methods. The student volunteers were normal healthy subjects.

In the case of the student subjects, an experimental pain was induced by a pressure cuff which had small plastic pyramids sewn into it. The cuff was placed around the upper arm and inflated at a constant rate. The subject was able to stop the pain at any time by releasing a pressure valve.

Melzack reported dramatic relief of pain in three patients with chronic back pain. The patients appeared much

calmer, visibly happier, and less anxious after the training procedures. The intensity of the pain was sharply reduced as reflected by responses on a questionnaire developed by Melzack. One patient reported she had reduced her intake of analgesics by fifty percent.

In a similar manner, the subjects who received experimental pain reported significant changes in perceived pain intensity after training and they were able to tolerate the pain for longer periods of time [Erickson, 1972].

In a subsequent study, Melzack [1972] tested subjects using parcellated procedures in order to determine the relative contributions of hypnosis and alpha training in the control of pathological pain. Basically, the study was designed with three major groups evaluated under the following conditions: (1) Hypnotic procedure plus feedback, (2) hypnotic procedure alone, and (3) alpha training alone. The subjects in each group were told that the procedure they were to receive would relieve their pain.

The clinical patients were again selected from a population that suffered severe chronic back pain or arthritic pain for several years. Many of the subjects took large amounts of analgesic drugs throughout the study.

The initial analysis of the data indicated that by themselves, neither the hypnotic suggestion nor the alpha training produced a significant reduction in pathological pain. However, under conditions of hypnotic procedure plus alpha training, there was a significant reduction in the

level of pain reported in a substantial number (64%) of the subjects. The alpha training alone had the smallest effect on the pain. The hypnotic suggestion had a larger effect but was not statistically significant.

In considering the results of Melzack's [1972] second study, it must be remembered that only chronic pain sufferers were tested. In the group receiving both hypnosis and alpha training, it was interesting to find that they were the only ones to achieve a significant increase in their alpha production.

Melzack infers from this study that alpha training by itself is ineffective in relieving pain. However, before accepting this conclusion, several factors regarding his second study must be considered. There was continued use of analgesics during the study with no reference to the intake levels. Only chronic pain sufferers were used as subjects. Pathological pain is almost always accompanied by high levels of anxiety and emotional strain [Smith, 1966]. This anxiety component must be considered an integral part of the pain experience when evaluating empirical findings [Sternbach, 1968]. Moreover, as was previously mentioned [Sternbach, 1968], hypnotic procedures enable a subject to greatly reduce his anxiety. Sternbach [1968] also states that it is the absence of anxiety concerning the noxious stimulation which is the single necessary and sufficient condition for perceiving the stimulus as a nonpainful sensation. Finally, as also previously indicated, extremely anxious people have been

shown to have difficulty producing alpha brain waves and relaxing [Budzinski and Stoyva, 1970]. In agreement with this finding, Green [1969] commented that his anxious subjects could not be trained to produce high alpha brain waves. "With time, the anxious subjects might learn to produce alpha but they would require a great deal more training than the normal individual."

From this perspective, the authors believe there is an explanation for the synergistic effect resulting from the combination of hypnosis and alpha training. Anxiety, present in nearly all pain sufferers, is highly effective in blocking alpha production. It is believed that the hypnotic suggestion effectively reduced anxiety to the level where alpha training could proceed, further relaxing the individual and thus reducing the pain.

Melzack's [1972] inference that alpha training alone is ineffective in reducing pain for the normal individual is brought into question.

II. EXPERIMENTAL DESIGN

A. PURPOSE OF THIS EXPERIMENT

The purpose of this experiment was to (1) replicate those studies that reported rapid alpha control using an alpha contingent auditory feedback signal, (2) replicate those studies that indicated alpha control would continue without further feedback, (3) investigate the effects of non-contingent and beta-contingent reinforcement of the control of alpha activity, and (4) investigate what effect the ability to control alpha has on a normal individual's tolerance to experimental pain and his anxiety level as indicated by the physiological measurements of blood pressure and pulse.

B. DEFINITION OF CONCEPTS

Ischemic Pain - Pain resulting from a temporary lack of blood supply in an organ or tissue.

Anxiety - A state of being uneasy, apprehensive or worried about what may happen. Anxiety usually results in elevated somatic activity. Physiological indicators, inter alia, include increased blood pressure, pulse rate, muscle tension and galvanic skin response. Psychological tests such as the Taylor Manifest Anxiety Scale, and the Multiple Affect Adjective Checklist also indicate the presence of Anxiety.

Alpha Control - The ability to increase or decrease alpha generation at will. Control is mathematically defined as the

difference in alpha levels of a subject when trying to generate alpha and when not trying to generate alpha.

C. HYPOTHESES

Hypothesis 1: Subjects receiving contingent alpha feedback training will show a significantly greater enhancement of alpha activity over their alpha basal level, than subjects receiving non-contingent or beta-contingent feedback training (under feedback conditions).

Hypothesis 2: Subjects receiving contingent alpha feedback training will show significantly greater enhancement of alpha activity over their alpha basal levels than subjects receiving non-contingent or beta-contingent feedback training (under no feedback conditions).

Hypothesis 3: Subjects receiving contingent alpha feedback training will show a significantly greater degree of alpha control, defined as the difference between alpha levels during the alpha on and alpha off sessions, than subjects receiving non-contingent or beta contingent feedback training (under feedback conditions).

Hypothesis 4: Subjects receiving contingent alpha feedback will show a significantly greater degree of alpha control, as defined above, than subjects receiving non-contingent or beta-contingent feedback training (under no feedback conditions).

Hypothesis 5: Subjects receiving contingent alpha feedback training will show a significantly greater tolerance to

an experimental pain than subjects receiving non-contingent or beta-contingent feedback training (under feedback conditions).

Hypothesis 6: Subjects receiving contingent alpha feedback training will show a significantly greater tolerance to an experimental pain than subjects receiving non-contingent or beta-contingent feedback training (under no feedback conditions).

Hypothesis 7: Regardless of the training subjects received, high alpha producers will show a significantly greater tolerance to an experimental pain than low alpha producers (under feedback conditions).

Hypothesis 8: Regardless of the training subjects received, high alpha producers will show a significantly greater tolerance to an experimental pain than low alpha producers (under no feedback conditions).

Hypothesis 9: Subjects receiving contingent alpha feedback training will show a significantly greater reduction in their systolic blood pressure during each training session than subjects receiving non-contingent or beta-contingent feedback training.

Hypothesis 10: Subjects receiving contingent alpha feedback training will show a significantly greater reduction in their diastolic blood pressure during each training session than subjects receiving non-contingent or beta-contingent feedback training.

Hypothesis 11: Subjects receiving contingent alpha feedback training will show a significantly greater reduction in their pulse rate during each training session than subjects receiving non-contingent or beta-contingent feedback training.

C. GENERAL DESIGN

In order to set the hypotheses and properly control for increases in alpha activity due simply to progressive relaxation and acclimatization to the experimental situation, three groups were established. The subjects in the first group (alpha group) received alpha-contingent binary auditory feedback. The subjects in the second group (yoked group) received non-contingent reinforcement through taped recordings of their "yoked" alpha partner's feedback signal. The subjects in the third group (beta group) received beta-contingent binary auditory feedback. However, the subjects in all three groups were told that the tone they heard indicated the presence of alpha which was activated by their own brain activity.

Presenting a comparable auditory feedback signal to the three groups also added a further control, in that, all subjects had a similar sounding tone to focus their attention on while their tolerance to an ischemic pain was measured.

Ischemic pain was used in this experiment for two reasons: (1) it has been demonstrated to be a satisfactory simulation of pathological pain [Smith et al., 1966] and therefore the potential extrapolation of laboratory research

to real life situations is increased, and (2) the subject can be exposed to the stimulus for several minutes, thus allowing time for cognitive mechanisms to operate.

D. SUBJECTS

Due to the substantial amount of time required for alpha control training sessions, a randomization of the sample could not be accomplished. Alternatively, all 15 subjects were volunteers with little or no meditative experience. The subjects were randomly assigned to the three groups with the only stipulation being that the three groups were statistically from the same population with respect to: (1) baseline percentage of alpha, and (2) basal tolerance to ischemic pain. The composition of the sample is as indicated in Table I.

E. APPARATUS

All alpha control training and pain tolerance treatments were conducted in a dimly lit, sound attenuated room (Industrial Acoustics Co., "Controlled Acoustical Environment Chamber"). The temperature ranged from 70°-75° F. Subjects were seated in a comfortable reclining chair. An intercom system allowed communication between subjects and experimenters.

1. Alpha Feedback Equipment

Alpha control training was conducted with an Aquarius Electronics' Alphaphone Brainwave Analyzer Model 1001DT. Complete technical details of this unit may be acquired by consulting the Brainwave Analyzer Instructional Manual.

Table I

Sample Composition

Subject	Sex	Age	Dominant Hand	Education
Alpha Group				
1	M	28	L	College
2	F	23	R	High School
3	M	29	R	College
4	M	28	L	College
5	M	28	R	College
Yoked Group				
1	M	26	R	College
2	M	22	R	Jr. College
3	M	30	L	College
4	F	25	R	High School
5	M	31	R	Jr. College
Beta Group				
1	M	38	R	Master's
2	M	35	R	College
3	M	30	R	Ph.D.
4	M	27	R	College
5	F	26	R	College

Cumulative alpha time was recorded to the nearest tenth of a second by the Aquarius Timer Model 1509 (See Appendix A). Binary feedback was used and consisted of a tone presented to the subject through a headphone set. Three flat, silver plated, surface electrodes on a 4-ft. shielded cable were used. For right-handed subjects, the electrodes were placed: (1) on the parietal lobe (position C₃); (2) on the occipital lobe (position O₁); and (3) on the left mastoid (ground position A₁). For left-handed subjects, the electrodes were placed: (1) on the parietal lobe (position C₄); (2) on the occipital lobe (position O₂); and on the right mastoid (ground position A₂). These position designators (A₁, O₂, etc.) are in accordance with the international (10-20) electrode placement (conventional) system.

2. Ischemic Pain Equipment

A standard adult-sized blood pressure cuff (Baumanometer Desk Model) was used to obstruct the flow of blood in the subject's arm. A Stoelting Co. hand dynamometer was utilized for the subject to squeeze (See Appendix A).

F. PROCEDURES

The training consisted of seven sessions for each subject. Each trraining session lasted approximately one hour. Tolerance to ischemic pain was measured on the first, fifth, and seventh session.

For the first session (Familiarization/FAM), the electrodes were attached as previously indicated and the

subject was then seated in a sound attenuated room. The subject's blood pressure and pulse were then measured. A tape recorded set of instructions was presented to the subject through a headphone set. The instructions consisted of a female voice describing the general design of the experiment and explaining what alpha control tasks the subject was to be involved in during the seven sessions. The instructions also described the methodology that was to be employed to induce ischemic pain but the word "pain" was not mentioned. Furthermore, there was no suggestion made that alpha training would be effective in diminishing the pain experienced. The taped instructions are presented verbatim in Appendix B.

The tape presentation was then followed by a 15-minute recording of the subject's basal alpha level. For this measurement, the subject was merely instructed to sit quietly with his eyes open. The subject was not given any feedback during this session. During the baseline measurements, the "noise threshold" was set at 20% for all subjects in accordance with the recommendations set forth in the Brainwave Analyzer Instruction Manual. The "noise threshold" adjusts the brainwave analyzer's noise suppression threshold. At its 0% position, the noise threshold is nearly zero. At its 100% position, the noise suppression circuit will require an input signal to exceed about 50 microvolts to activate the feedback tone. Otherwise, the input signal will be considered as noise vice alpha brainwave activity. For most training

purposes, the manufacturers recommended the "noise threshold" be positioned at a minimum of 20% (about 10 microvolts) to filter out slight eye movements which might otherwise mimic brainwave activity.

The alpha baseline measurement was immediately followed by the tolerance measurement to ischemic pain for the right arm (PT/RA). For this test, the Modified Submaximum Effort Tourniquet Technique [Smith et al., 1966; Johnson, 1973] was employed. The procedure varied from that used by Smith et al. [1966] in that the arm was not exsanguinated by elevating it and applying an Esmarch bandage. The subject was seated with his forearm resting comfortably on the chair's armrest. A standard adult-sized blood pressure cuff was applied to his upper arm and inflated and maintained at a pressure of 250 millimeters of mercury. Alpha measurements commenced upon cuff inflation, but again the subject was not given any feedback signal. The subject then squeezed the hand dynamometer 20 times. The 10 kilogram point on the dynamometer was marked and the subject was instructed to squeeze just to the mark. Each repetition was timed to last 2 seconds, followed by a 2 second pause. The exercise schedule was presented to the subject via tape recorded orders consisting of "squeeze", "hold", and "release". The dial and bulb for inflation of the cuff were outside the chamber, separated from the subject by 8-feet of rubber tubing. This allowed the experimenters to monitor the pressure in the cuff without distracting the subject with their presence. The

subject was instructed to verbally report on the sensations in his forearm by using a scale from 0 to 4. Zero indicated no distress, one indicated slight distress, two indicated moderate distress, three indicated a very distressing condition, and four indicated that point at which the subject very much wished to have the cuff removed. The subject was instructed to simply call out that number which represented the best description of his sensations as they occurred. The experimenters timed his calls outside the chamber and released the pressure when the subject reported "four". The verbal scale was also posted on the chamber's wall in clear view of the subject.

After a brief rest to allow the subject's right arm to return to a normal condition, the technique was repeated for his left arm (PT/LA).

At the completion of the tolerance measure on the left arm and while the subject was still comfortably seated, his blood pressure and pulse were again recorded. (Due to equipment limitations, blood pressure and pulse were not constantly monitored but were measured at the beginning and end of each session.) The subject was then removed from the chamber, the electrodes were detached and the subject completed a short questionnaire pertaining to his physical activities, feelings and impressions for that day.

When all subjects had completed their first session (FAM), they were then randomly assigned to the three groups. The previously mentioned statistical stipulation of group

composition, with respect to basal alpha and tolerance to ischemic pain, was based on the results of the FAM session.

Alpha control training began with the Training Session One (TS 1). The method of alpha control training employed in this experiment was adapted from a study by Hord and Barber [1971]. The procedure varied from their study, in that, each training session consisted of eight 5-minute tasks vice eight 8-minute tasks, and the order in which each task was presented varied from one training session to the next, vice having the same task appearing in the same order each training session. Additionally, Hord and Barber [1971] only conducted alpha control training for two days.

In TS-1, the EEG electrodes were attached as before and the subject was again seated in the sound attenuated chamber. Before each training session there was a brief familiarization session in which, the experimenters asked the subject to indicate a volume and tone preference for the auditory feedback signal. This familiarization session also afforded the experimenters time to make a trial-and-error adjustment of the "noise threshold" so that the subject's alpha waves would enable the feedback tone approximately 30% of the time. Thus, it was the experimenters' intentions to try to equalize across the subjects, the "alpha basal level," independent of each subjects absolute abundance of alpha.

Each 5-minute task was initiated by a brief explanation and appropriate set of instructions via intercom from the experimenters. Training Session 1 (TS-1) was conducted as follows:

1. Baseline (BL). "You are instructed to sit quietly with your eyes open for awhile."
2. Alpha on, with feedback (ON,FB). "You are instructed to keep alpha on as much as possible, using the contingent feedback tone as a guide. Maintain eyes open."
3. Alpha on, no feedback (ON,NFB). "You are instructed to try to produce alpha activity on the basis of whatever understanding you might now have about alpha. Maintain eyes open."
4. Alpha off, with feedback (OFF,FB). "You are instructed to keep alpha off as much as possible using the contingent feedback tone as a guide. Maintain eyes open."
5. Alpha off, no feedback (OFF, NFB). "You are instructed to keep alpha off, on the basis of whatever understanding you might now have about alpha. Maintain eyes open."
6. Alpha on, with feedback (OB,FB). Instructions were the same as task #2.
7. Alpha on, no feedback (ON,NFB). Instructions were the same as task #3.
8. Recovery (REC). Instructions were the same as task #1.

Cumulative alpha time was read to the nearest tenth of a second from the Aquarius Timer for each 5-minute task. The percentage of alpha for each 5-minute task was then computed. (After each task, the timer was reset to zero and a cumulative record for the next 5-minute task was started.)

Training session two (TS-2), training session three (TS-3) and training session four (TS-4) were conducted exactly the same as TS-1 except that the order in which each task was presented varied in the different sessions. See Appendix C for the overall training session design.

As previously mentioned the tolerance measure to ischemic pain was performed again in the fifth and seventh sessions. These sessions are denoted in Appendix B as Pain Tolerance One (PT-1) and Pain Tolerance Two (PT-2). For these sessions the EEG electrodes were again attached in the same manner, and the Modified Submaximum Tourniquet technique [Smith et. al., 1966; Johnsen 1973], as outlined for the FAM session, was performed in both these sessions.

Again each task was initiated by a brief explanation and appropriate set of instructions via intercom from the experimenters. PT-1 and PT-2 were conducted as follows:

1. Baseline (BL). The instructions remained the same.
2. Alpha on, with feedback (ON,FB). The instructions remained the same.
3. Alpha on, with no feedback (ON,NFB). The instructions remained the same.
4. Pain Tolerance Right Arm with Feedback (PT/RA/FB). The blood pressure cuff was applied to the right arm. The subject was instructed to verbally report on the sensation in his forearm using the same scale from 0 to 4. Additionally, he was instructed to keep "alpha" on as much as possible, using the contingent feedback tone as a guide with his eyes open.

5. Pain Tolerance Left Arm without Feedback (PT/LA/NFB) .

The blood pressure cuff was transferred to the left arm. Again, the subject was instructed to verbally report on the sensations in his forearm using the same scale from 0 to 4. Additionally, he was instructed to try to produce alpha activity on the basis of whatever understanding he might then have of alpha with his eyes open.

6. Recovery (REC). The instructions were the same as baseline.

III. RESULTS

A. ALPHA FEEDBACK TRAINING

The alpha control training performance of the three groups is presented in Appendix D, Table I. The figures appearing in the table under category BL, represent an average % of alpha activity for BL and recovery tasks in each session. Likewise, in those sessions where there was a duplication of tasks, that is, for tasks ON,FB and ON,NFB; the duplicated tasks were combined and the percentage figure for alpha, as it appears in the table, represents an average % of alpha for those two tasks.

The alpha training performances of the three groups are summarized and presented in Table I as group average % alpha activity for the different tasks.

An F-test comparison of the basal alpha measures of the three groups for the Familiarization Session was performed to determine if there was a sampling error made initially. The F-test revealed that there was no statistically significant difference between the three groups with respect to alpha baseline measurements ($F = .194$, $df = 2,12$).

For Hypothesis 1, the percent alpha differences between BL and ON,FB for the three groups are illustrated in Figure 1. The figure indicates that the alpha group generally had greater differences between % alpha emitted during BL tasks and when they were instructed to produce alpha with feedback

than the other two groups. The figure also indicates that the yoked control group had a greater alpha % difference than the beta control group.

An analysis of variance, two-factor mixed design: repeated measures on one factor [Bruning and Kintz, 1968] was used to: (1) compare the overall performance of the three groups, (2) evaluate performance changes over trials, and (3) evaluate the different group training effects in relation to the passage of the time between trials. The analysis presented in Table II showed the group training main effect was significant at the .10 level. The trials main effect was significant at the .05 level. The group trials interaction was not significant (NS).

The Duncan Range Test revealed that the alpha group and the yoked control group did not differ significantly in their overall performance. However, both the alpha group and the yoked group did differ significantly from the beta control group ($p < .01$). An F-test for simple effects revealed that only the performance of the beta group did change significantly as a function of trials, ($p < .10$). These results indicated that the subjects who received relevant reinforcement (alpha group) and intermittent reinforcement (yoked group) performed better in enhancing their alpha activity over their basal level than subjects who received relevant reinforcement contingent upon beta brainwave activity.

For Hypothesis 2, the percent alpha differences between BL and ON,NFB for the three groups are illustrated in Figure 2. Again, the figure indicates that the alpha group generally had greater differences between % alpha emitted during BL tasks and when they were involved in an alpha producing task without feedback. Additionally, the figure indicates that the yoked control group had a greater % alpha difference between the two tasks than the beta control group.

The same analysis of variance as mentioned for Hypothesis 1 was performed. The analysis presented in Table III showed the group training main effect was significant at the .025 level. Neither the trials main effect nor the group-trials interaction was significant.

The Duncan Range Test revealed that the alpha group and the yoked control group did not differ significantly in their overall performance. However, both the alpha group and the yoked group did differ significantly from the beta control group ($p < .01$). These results indicated that the subjects who received relevant reinforcement (alpha group) and intermittent reinforcement (yoked group) performed better in enhancing their alpha activity over their basal level with no feedback than subjects who had received relevant reinforcement contingent upon beta brainwave activity.

With respect to Hypothesis 3, the percent alpha differences between ON,FB and OFF, FB for the three groups are illustrated in Figure 3. Figure 3 indicates that the alpha group had greater differences than the control groups between

% alpha produced during ON,FB and OFF,FB tasks. Likewise, the figure shows that the yoked group achieved greater differences in alpha % during ON,FB tasks and OFF,FB tasks than the beta group.

An analysis of variance, presented in Table IV, revealed a group main effect significant at the .025 level. Neither the trials main effect nor the group-trials interaction was significant.

The Duncan Range test revealed that the alpha group differed significantly in their performance from the yoked group ($p < .01$), and the beta group ($p < .01$). The test further showed that the yoke group differed significantly from the beta group ($p < .01$). These results confirm hypothesis three that the subjects receiving contingent alpha feedback training will show a greater degree of alpha control than subjects receiving noncontingent or beta contingent feedback training.

For Hypothesis 4, the percent alpha differences between ON,NFB and OFF, NFB for the three groups are illustrated in Figure 4. This figure again shows that the alpha group had greater differences between % alpha produced during ON,NFB tasks and OFF,NFB tasks than the other two control groups. Additionally, the figure shows that the yoked group also had greater differences in alpha % during ON,NFB tasks and OFF,NFB tasks than the beta group.

An analysis of variance, presented in Table VI, found a group main effect significant at the .01 level. Again,

neither the trials main effect nor the group-trials interaction was significant.

The Duncan Range Test showed that the alpha group differed significantly in alpha control ability from the yoked group ($p < .01$), and the beta control group ($p < .01$). It further revealed that the yoked group differed significantly from the beta group in alpha control ability ($p < .01$). Thus hypothesis four is confirmed.

B. TOLERANCE TO ISCHEMIC PAIN

The pain tolerance performances of the three groups is presented in Appendix D, Table II. The figures appearing in Table II under the categories "PT/RA" and "PT/LA" represent the total seconds the subject endured the inflated pressure cuff for the right arm and left arm respectively. The figures appearing in Table II under the categories "Alpha %, PT/RA and PT/LA" represent the subject's percentage of alpha produced while the inflated pressure remained on the right and left arms.

The pain tolerance performances of the three groups are summarized and presented in Table II as the average time (secs.) each group endured the inflated pressure cuff. Additionally, each groups' average % of alpha activity produced while the cuff was inflated appears in Table II.

An F-test comparison of the basal pain tolerances of the three groups for the Familiarization Session was performed to determine if there was a sampling error made

initially. The F-test revealed that there was no statistically significant difference between the three groups with respect to their initial right arm tolerance to ischemic pain ($F = .16$, $df = 2,12$). The F-test also revealed that there was no statistically significant difference between the three groups with respect to their initial left arm tolerance to ischemic pain ($F = .07$, $df = 2,12$).

For Hypothesis 5, the ischemic pain tolerance performances under feedback conditions for the three groups are illustrated in Figure 5. The figure indicates that the alpha group displayed a greater tolerance to ischemic pain than both the yoked and beta groups. Moreover, it appears that the alpha group increased its tolerance to ischemic pain at a greater rate over the sessions than the two other control groups.

The same analysis of variance that was previously described was performed. The analysis presented in Table VI, found no significant group main effect or group X trials interaction. However, the trials main effect was found to be significant at the .05 level. Applying an F-test for simple effects, it was found that only the pain tolerance of the alpha group did increase as a function of trials, ($p < .10$).

These results, although not completely confirming Hypothesis 5 because of no overall significant difference between groups, support the hypothesis to the extent that only the group that received alpha contingent feedback showed a

statistically significant increased tolerance to ischemic pain over trials.

With respect to Hypothesis 6, the ischemic pain tolerance performances under no feedback conditions for the three groups are illustrated in Figure 6. This figure again indicates that the alpha group generally displayed a greater tolerance to ischemic pain than both control groups. It also shows that only the alpha group continually increased its tolerance to ischemic pain.

However, the analysis of variance as presented in Table VII, found no significant group main effect, nor group-trials interaction. The trials main effect was found to be significant only at the .20 level. Again, applying an F-test for simple effects, it was revealed that only the alpha group significantly increased its tolerance to ischemic pain as a function of trials ($p < .10$).

As with Hypothesis 5, these results do not completely confirm Hypothesis 6 because of no overall significant difference between groups. However, the hypothesis is supported when one considers that only the group that received alpha contingent feedback showed a statistically significant increased tolerance to ischemic pain over trials.

For Hypothesis 7, the subjects were divided into two groups based on their alpha production with the cuff inflated under feedback conditions. The High Alpha group per session consisted of those seven subjects who exceeded the overall

session median percentage of alpha produced for each of the three sessions in which pain tolerance was measured. The Low Alpha group per session consisted of those seven subjects who produced less than the overall session median percentage of alpha for each of the three sessions in which pain tolerance was measured. The pain tolerance measure of the median subject for each of the three sessions was excluded in order to equalize the number of subjects in both groups. The pain tolerance performances of the two groups under both feedback (PT/RA) and no feedback (PT/LA) conditions are summarized and presented in Table X as the average time (secs.) that each group endured the cuff. In addition, both groups' average percentage of alpha activity produced during the pain tolerance measurement appears in Table X.

The ischemic pain tolerance performances under feedback conditions for the two groups are presented in Figure 7. The figure indicates that, except for the FAM session, the High Alpha group displayed a greater tolerance to ischemic pain than the Low Alpha group. However, the analysis of variance as presented in Table VIII found the group main effect to be significant only at the .20 level. The trials main effect and group trials interaction were not significant. Additionally, the Duncan Range test found no significant difference at .10 level in terms of the two groups overall performance.

For Hypothesis 8, the subjects were divided into two groups based on their alpha production with the cuff inflated

under no-feedback conditions ("Alpha %, PT/LA"). The procedures to establish the two groups for each session were identical to those described for Hypothesis 7, except the percentage of alpha considered was that produced without feedback.

The ischemic pain tolerance performances under no-feedback conditions for the two groups are presented in Figure 8. The figure indicates that the High Alpha group consistently displayed a greater tolerance to ischemic pain than the Low Alpha group.

The analysis of variance as presented in Table IX revealed a group main effect significant at the .025 level. Neither the trials main effect nor the group trials interaction was significant.

The Duncan Range Test showed that the High Alpha group differed significantly from the Low Alpha group in their overall tolerance to ischemic pain when no feedback tone was present ($p < .001$). Thus Hypothesis 8 is confirmed.

C. ANXIETY REDUCTION (PHYSIOLOGICAL INDICES)

The blood pressure and pulse rate measurements of the three groups are presented in Appendix D, Table IV. As previously mentioned, due to equipment limitations, each subject's blood pressure and pulse could not be continuously monitored throughout the session but had to be measured by standard medical examining techniques at the beginning and end of each session. The figures appearing in Appendix D under the categories "BPB" and "BPA" represent each subjects

systolic/diastolic blood pressure "Before" and "After" each session respectively. Likewise, the figures appearing in Table III under the categories "PB" and "PA", represent each subject's pulse "Before" and "After" each session respectively.

In testing Hypotheses 9, 10, and 11 only the Training Sessions (TS1, TS2, TS3, and TS4) were considered in evaluation of the three groups' reduction of blood pressures and pulse rates. This was necessary because of the previously mentioned equipment limitations which did not permit continuous monitoring of the subjects' physiological responses. Only in these four sessions did each subject spend exactly the same amount of time in the experimental chamber. For sessions FAM, PT1, and PT2, the amount of time each subject spent in the chamber was contingent upon the length of time he chose to tolerate the inflated pressure cuff. Consequently, during those three sessions there was no way to equalize across the subjects a time dependent measurement of somatic activity.

The physiological measures of the three groups are summarized and presented in Appendix D, Table III as the group average blood pressure and pulse rate existing before and after each relevant session.

For Hypothesis 9, the three groups' systolic blood pressure changes for the four training sessions are illustrated in Figure 9. In considering each subject's systolic blood pressure change, a reduction in the millimeters of mercury

was considered a positive performance and conversely an increase in the millimeters of mercury a negative performance. This experimental determination is reflected in Figure 9. Figure 9 indicates that the alpha group generally displayed a greater reduction in its systolic blood pressure than both control groups. Only in TS1 did the yoke group display a greater reduction in its systolic blood pressure than the alpha group. Figure 9 also indicates that the yoke group consistently produced a greater reduction in its systolic blood pressure than the beta group.

The analysis of variance presented in Table XI found a group main effect significant at the .001 level. The trials main effect was only significant at the .20 level. The group trials interaction was not significant.

The Duncan Range Test revealed that the alpha and yoke group did not differ significantly in their overall performance. However, both the alpha and the yoke group did differ significantly in their overall performance from the beta control group ($p < .01$).

Applying an F-test for simple effects, it was found that only the alpha group's reduction in systolic blood pressure showed a significantly greater reduction during each session as the sessions progressed ($p < .10$).

These results indicated that the subjects who received relevant alpha reinforcement (alpha group) and intermittent alpha reinforcement (yoke group) performed better in reducing their systolic blood pressure than subjects who received relevant reinforcement contingent upon beta brainwave activity.

For Hypothesis 10, the three groups' diastolic blood pressure changes for the four training periods are illustrated in Figure 10. Again, a reduction in diastolic blood pressure was considered a positive performance and an increase in diastolic blood pressure a negative performance. The results are reflected in Figure 10. From Figure 10, except for TS3, the alpha group displayed a greater reduction in its diastolic blood pressure than the two control groups. The yoked group also displayed a slightly greater reduction in its diastolic blood pressure than the beta control group.

However, the analysis of variance presented in Table XII found no significant group main effect, trials main effect, nor group-trials interaction.

For Hypothesis 11, the three groups' pulse rate changes for the four training sessions are illustrated in Figure 11. Again, a reduced pulse rate was considered a positive performance and, conversely, an increased pulse rate a negative performance. The results are reflected in Figure 11. Figure 11 indicates that the alpha and yoke group did not differ much in their overall performance in reducing their pulse rates. However, from Figure 11 it appears that both the alpha and yoke group performed better at reducing their pulse rates than the beta group.

The analysis of variance presented in Table XIII found a group main effect significant at the .10 level. Neither

the trials main effect nor the group-trials interaction were significant.

The Duncan Range Test revealed that the alpha and yoke group did not differ significantly in their overall performance. However, consistent with Figure 11, both the alpha and the yoke group differed significantly in their overall pulse reduction from the beta group ($p < .01$).

These results indicated that the subjects who received relevant alpha reinforcement (alpha group) and intermittent alpha reinforcement (yoke group) performed significantly better in reducing their pulse rate than subjects who received relevant reinforcement contingent upon beta brain-wave activity.

Table II
Analysis of Variance for Alpha Enhancement
Over Baseline Activity With Feedback

Source	SS	df	ms	F	p
Total	17,964.3	44	--	--	--
Between subjects	9,635.9	14	--	--	--
Conditions	3,620.4	2	1810.2	3.61	< .10
Error	6,015.5	12	501.3	--	--
Within subjects	8,328.4	30	--	--	--
Trials	1,721.1	2	860.5	3.43	< .05
Trials X conditions	590.6	4	147.7	.58	NS
Error	6,016.8	24	250.7	--	--

Table III
Analysis of Variance for Alpha Enhancement
Over Baseline Activity Without Feedback

Source	SS	df	ms	F	p
Total	12,654.8	44	--	--	--
Between subjects	7,544.9	14	--	--	--
Conditions	4,008.1	2	2004.0	6.80	< .025
Error	3,536.8	12	294.7	--	--
Within subjects	5,110.0	30	--	--	--
Trials	150.1	2	75.0	.43	NS
Trials X conditions	813.6	4	203.4	1.177	NS
Error	4,146.3	24	172.8	--	--

Table IV
Analysis of Variance for Alpha-Control
Under Feedback Conditions

Source	SS	df	ms	F	p
Total	6,853.9	59	--	--	--
Between subjects	2,841.6	14	--	--	--
Conditions	1,320.6	2	660.3	5.21	< .025
Error	1,521.0	12	126.8	--	--
Within subjects	4,012.3	45	--	--	--
Trials	237.8	3	79.3	.808	NS
Trials X conditions	245.4	6	40.9	.417	NS
Error	3,529.2	36	98.0	--	--

Table V
An Analysis of Variance for Alpha-Control
Under No-Feedback Conditions

Source	SS	df	ms	F	p
Total	5,970.4	59	--	--	--
Between subjects	2,668.1	14	--	--	--
Conditions	1,527.9	2	764.0	8.04	< .01
Error	1,140.2	12	95.0	--	--
Within subjects	3,302.3	45	--	--	--
Trials	215.7	3	71.9	1.08	NS
Trials X conditions	697.2	6	116.2	1.75	NS
Error	2,389.3	36	66.4	--	--

Table VI
Analysis of Variance Pain Tolerance
With Feedback

Source	SS	df	ms	F	p
Total	1,877,718.0	44	--	--	--
Between subjects	1,322,070.0	14	--	--	--
Conditions	135,217.9	2	67,609.0	.684	NS
Error	1,186,852.1	12	98,904.3	--	--
Within subjects	555,648.0	30	--	--	--
Trials	131,443.6	2	65,721.8	3.90	< .05
Trials X conditions	19,424.8	4	4,856.2	.288	NS
Error	404,779.6	24	16,865.8	--	--

Table VII
Analysis of Variance Pain Tolerance
No Feedback

Source	SS	df	ms	F	p
Total	1,975,285.0	44	--	--	--
Between subjects	1,684,356.0	14	--	--	--
Conditions	45,366.2	2	22,683.1	.166	NS
Error	1,683,989.9	12	136,582.5	--	--
Within subjects	290,929.0	30	--	--	--
Trials	41,393.6	2	20,696.8	2.45	< .20
Trials X conditions	47,164.3	4	11,791.1	1.40	NS
Error	202,371.2	24	8,432.1	--	--

Table VIII
 Analysis of Variance Pain Tolerance
 High VS Low Alpha Producers with Feedback

Source	SS	df	ms	F	p
Total	1,703,286.4	41	--	--	--
Between subjects	733,907.3	13	--	--	--
Conditions	144,672.5	1	144,672	2.95	< .20
Error	589,234.8	12	49,103	--	--
Within subjects	969,379.1	28	--	--	--
Trials	96,417.6	2	48,208.8	1.44	NS
Trials X conditions	73,217.2	2	36,608.6	1.10	NS
Error	799,744.3	24	33,322.7	--	--

Table IX
 Analysis of Variance Pain Tolerance
 High VS Low Alpha Producers without Feedback

Source	SS	df	ms	F	p
Total	1,875,438.0	41	--	--	--
Between subjects	742,110.5	13	--	--	--
Conditions	282,080.1	1	282,080.1	7.36	< .025
Error	460,030.4	12	38,335.9	--	--
Within subjects	1,133,327.5	28	--	--	--
Trials	58,642.0	2	29,321.2	.65	NS
Trials X conditions	7,937.8	2	3,968.9	.89	NS
Error	1,066,747.7	24	44,447.8	--	--

Table X
 Pain Tolerance Performance
 For High and Low Alpha Groups

Familiarization				
Group	PT/RA	PT/LA	PT/RA	* Alpha % PT/LA
High Alpha	336 secs	425 secs	34.7%	49.5%
Low Alpha	312	262	21.8	28.7
Pain Tolerance One				
Group	PT/RA	PT/LA	PT/RA	* Alpha % PT/LA
High Alpha	478 secs	516 secs	49.2	53.7
Low Alpha	271	313	28.7	29.7
Pain Tolerance Two				
Group	PT/RA	PT/LA	PT/RA	* Alpha % PT/LA
High Alpha	501 secs	403 secs	56.6%	55.3%
Low Alpha	367	278	30.4	32.0

* Indicates % of alpha during pain tolerance measurement

Table XI
Analysis of Variance
Systolic Blood Pressure

Source	SS	df	ms	F	p
Total	3,218.6	59	--	--	--
Between subjects	1,227.0	14	--	--	--
Conditions	922.0	2	461.0	15.58	< .001
Error	355.0	12	29.6	--	--
Within subjects	1,941.6	45	--	--	--
Trials	212.1	3	70.7	1.8	< .20
Trials X conditions	314.1	6	52.4	1.33	NS
Error	1,415.4	36	39.3	--	--

Table XII
Analysis of Variance
Diastolic Blood Pressure

Source	SS	df	ms	F	p
Total	1,622.2	59	--	--	--
Between subjects	629.9	14	--	--	--
Conditions	112.6	2	56.3	1.31	NS
Error	517.3	12	43.1	--	--
Within subjects	992.3	45	--	--	--
Trials	65.3	3	21.8	.99	NS
Trials X conditions	141.9	6	23.7	1.08	NS
Error	785.1	36	21.9	--	--

Table XIII
Analysis of Variance
Pulse Rate

Source	SS	df	ms	F	p
Total	1,645.9	59	--	--	--
Between subjects	561.1	14	--	--	--
Conditions	202.5	2	101.3	3.39	< .10
Error	358.6	12	29.9	--	--
Within subjects	1,084.8	45	--	--	--
Trials	104.1	3	34.7	1.40	NS
Trials X conditions	87.3	6	14.6	.59	NS
Error	893.4	36	24.8	--	--

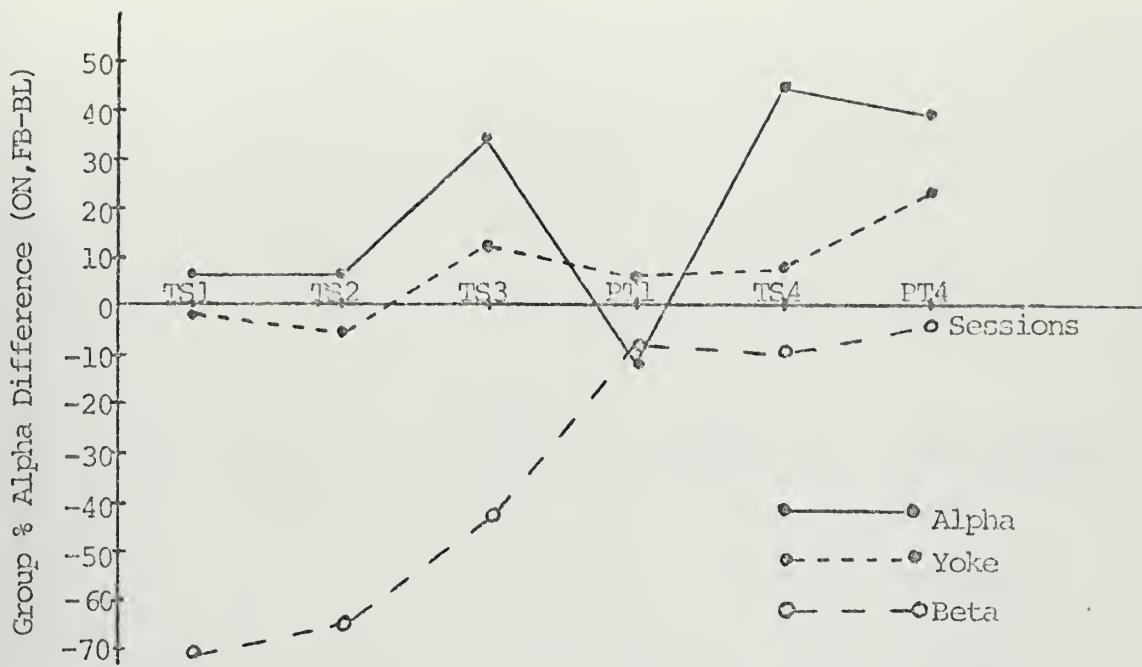


Figure 1: Alpha Enhancement Over Alpha Basal Levels with Feedback

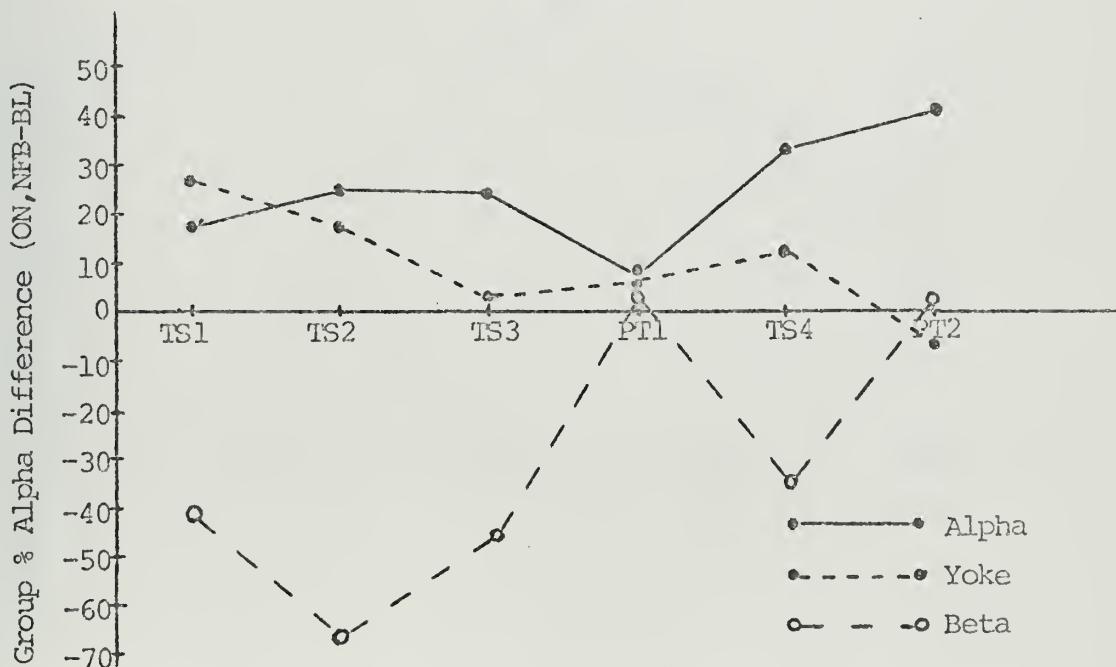


Figure 2: Alpha Enhancement Over Alpha Basal Levels without Feedback

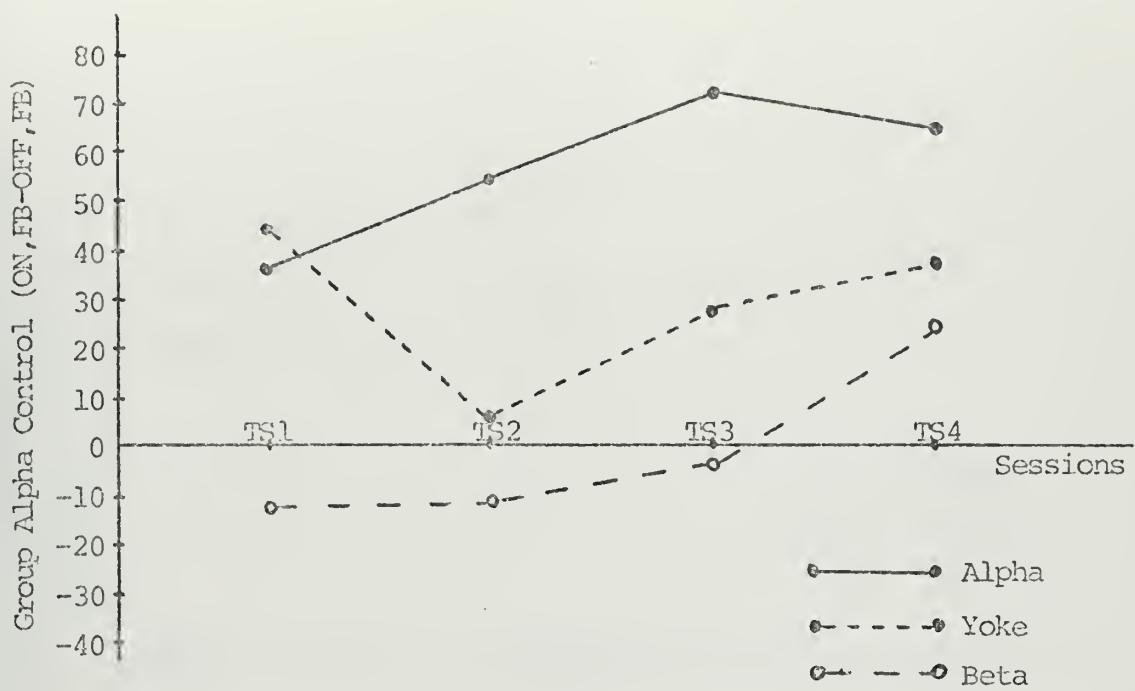


Figure 3: Alpha Control of Subjects with Feedback

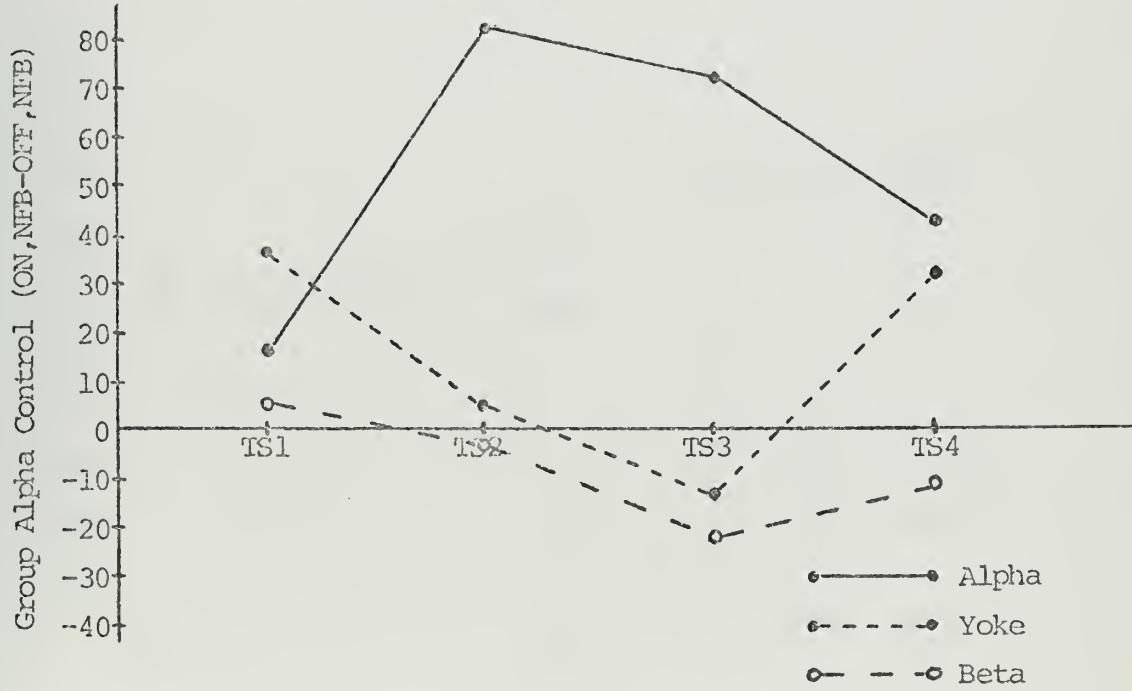


Figure 4: Alpha Control of Subjects without Feedback

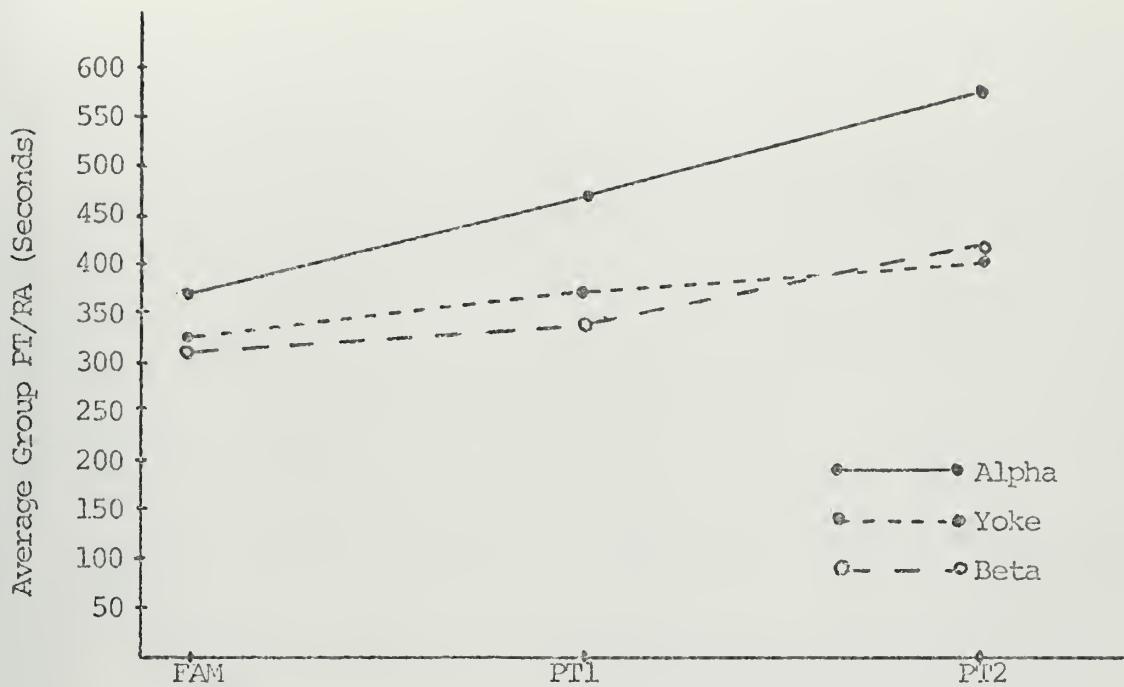


Figure 5: Ischemic Pain Tolerance with Feedback in PT1 and PT2

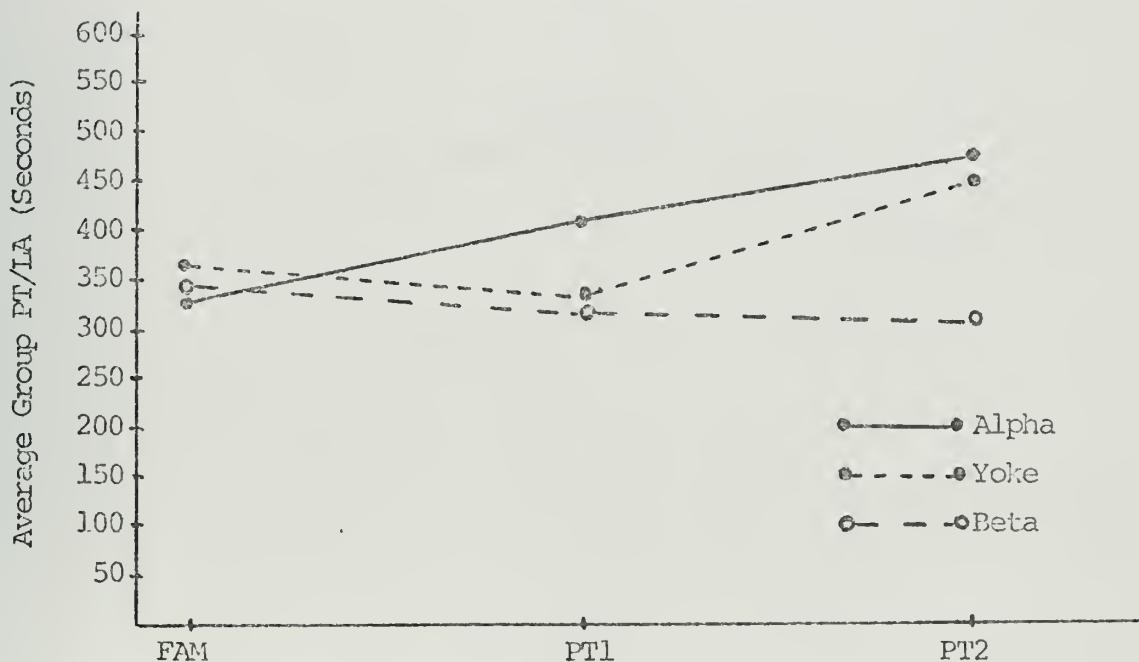


Figure 6: Ischemic Pain Tolerance without Feedback in PT1 and PT2

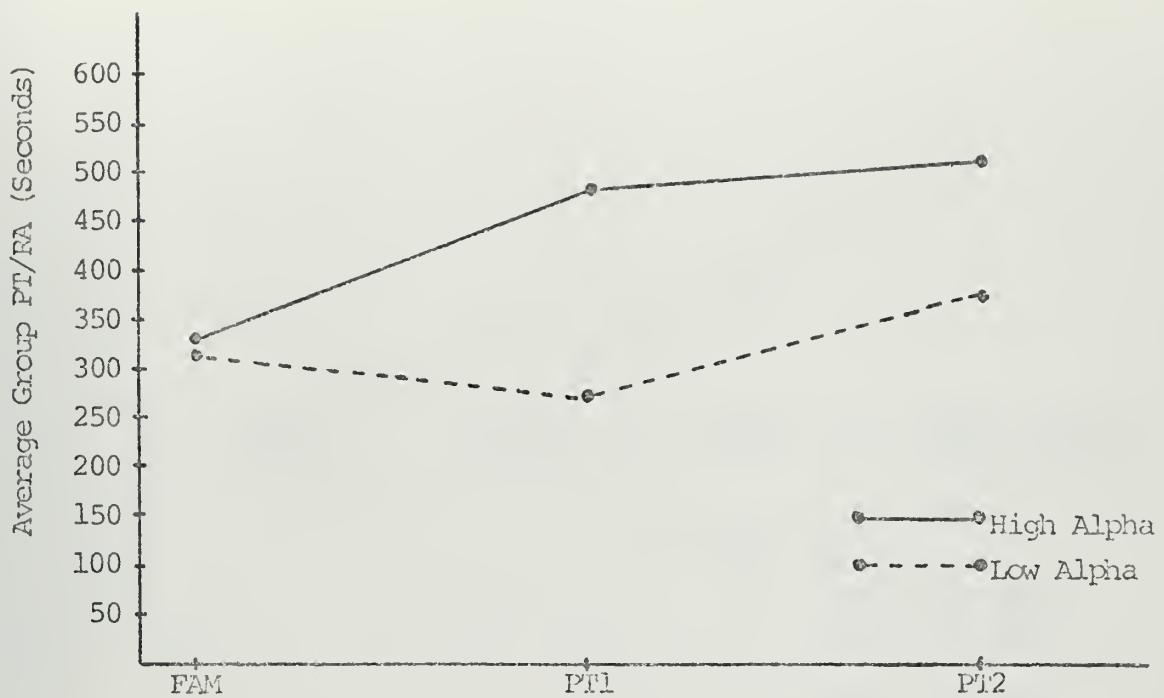


Figure 7: Ischemic Pain Tolerance, High vs. Low Alpha Producers with Feedback in PT1 and PT2

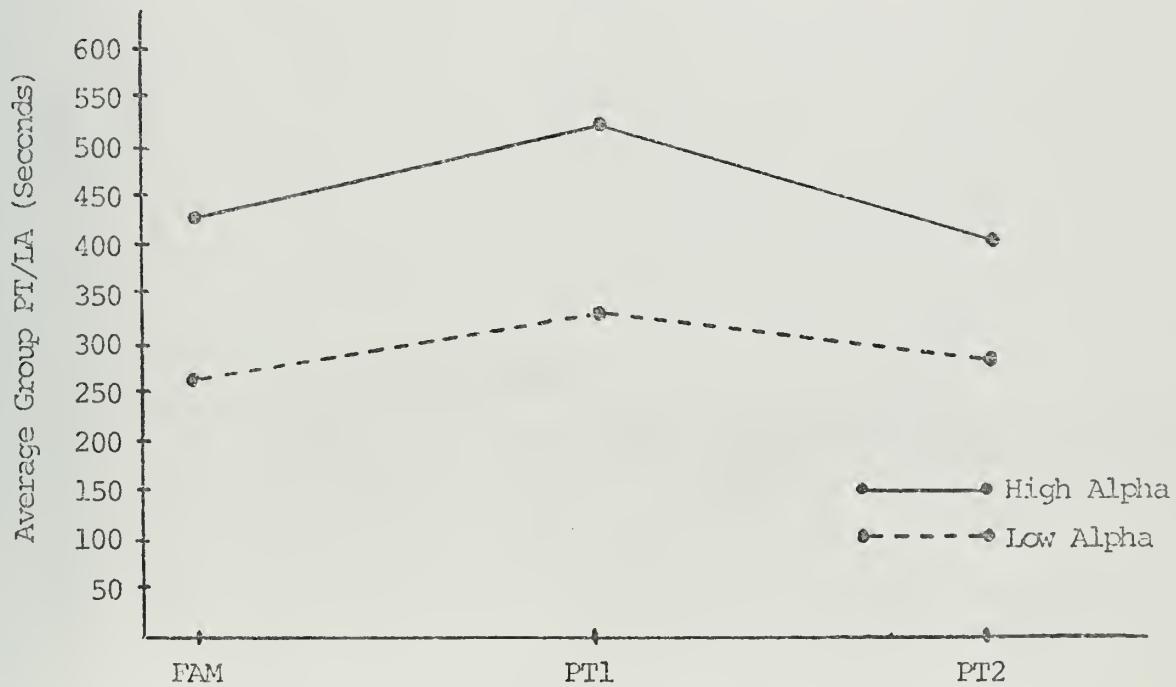


Figure 8: Ischemic Pain Tolerance, High vs. Low Alpha Producers without Feedback in PT1 and PT2

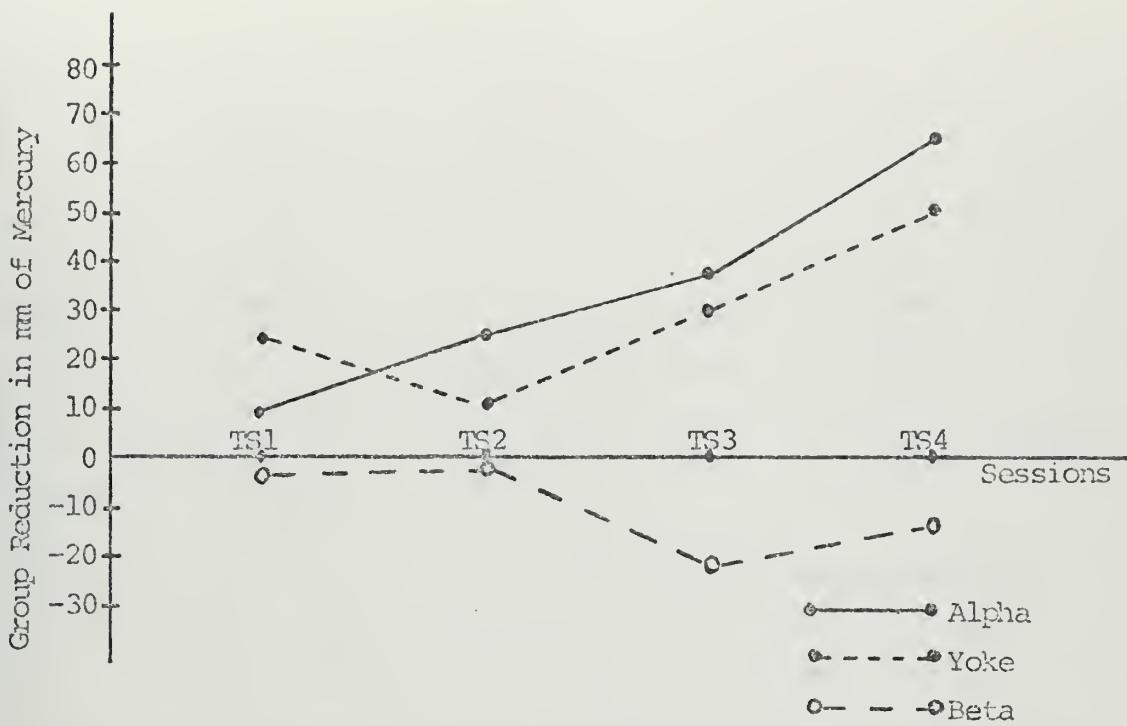


Figure 9: Reduction of Subjects' Systolic Blood Pressure within Training Sessions

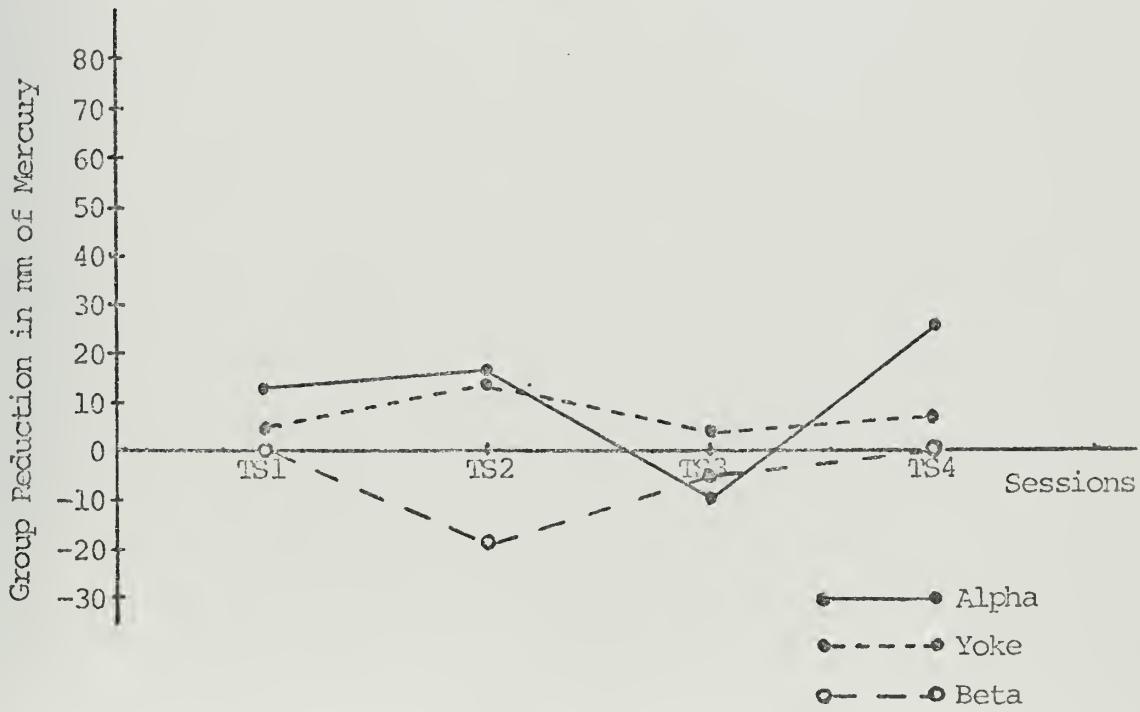


Figure 10: Reduction of Subjects' Diastolic Blood Pressure within Training Sessions



Figure 11: Reduction of Subjects' Pulse within Training Sessions

IV. DISCUSSION

A. ALPHA FEEDBACK TRAINING

For Hypotheses 1 and 2, the finding that there was no significant difference between the alpha and yoked group's enhancement of alpha over their baseline measures supports the results of Cleeland et. al. [1971]. Cleeland found no significant differences in the amount of alpha generated by contingently reinforced and yoked control subjects at the end of binary feedback training.

However, it is important to note that both the alpha group and the yoked group were significantly different from the beta contingent group. This finding is in accord with the report of Travis et. al. [1974], who found that their yoked group significantly outperformed their no-reinforcement group in increased output of alpha activity. As Travis et. al. [1974] hypothesized, this difference may have resulted because the yoked control subjects presumably attempted to comply with the same instructions as the experimental subjects, but received relevant reinforcement only when their alpha production coincided with that of the experimental group. At all other times, the yoked group was receiving erroneous (i.e. tone without alpha or alpha without tone) and random feedback. On the other hand, the beta group was receiving contingent, negative reinforcement. The effect of intermittent reinforcement needs further investigation to clarify

its effects on the alpha-producing response. The results of the present study indicate that subjects given relevant reinforcement and intermittent reinforcement perform better at enhancing their alpha activity over their basal level than subjects given relevant reinforcement contingent upon beta brainwave activity under feedback and no-feedback conditions.

The concept of "alpha control" is basic to alpha training. It is a strong indicator that a subject truly knows the alpha state and has achieved a degree of volitional control over it. With respect to Hypotheses 3 and 4, the finding that there was a significant difference between all three of the groups' performance at alpha control, under feedback and no feedback conditions, lends support to the belief that volitional control over the alpha brainwave can be achieved through operant conditioning techniques. The results of the present study regarding "alpha control" are in accord with the findings of Dewan [1966], Kamiya [1969], Nowlis and Kamiya [1970], Hord and Barber [1971], and Brown [1974]. The finding that the yoked group outperformed the beta group in alpha control, again, possibly indicates the effects of intermittent reinforcement. Several subjects in the control groups made statements to the effect that they didn't really feel like they had good "control" over the tone; especially when the task involved turning it off. The present results support their contention that they did not really learn the precise "feeling" of alpha; at least not as well as the experimental group.

B. TOLERANCE TO ISCHEMIC PAIN

Hypotheses 5 and 6 are not confirmed by the results of the present study. The results indicate no significant difference in tolerance to an experimental pain between subjects receiving contingent alpha feedback training and the two control groups, either with feedback or without feedback. The insignificant differences between the three groups tolerance to an experimental pain support the findings of Melzack [1972] who found that the alpha training procedures alone did not produce a significant reduction in clinical pain.

However, in considering tolerance to ischemic pain by groups, the experimenters think it warrants mention that only the alpha group displayed any significant increase in their pain tolerance over trials. Although hypotheses 5 and 6 cannot be confirmed with an F-test for simple effects significant at the .10 level, it is possible that this test may indicate a trend.

This training design only involved four sessions totally devoted to alpha control training. The two sessions (PT1 and PT2) wherein pain tolerance was measured subsequent to the FAM session, included only ten minutes of alpha enhancement time before the pressure cuff was applied.

In this study the experimenters purposely avoided any suggestions that alpha training procedures would effectively diminish experimental pain. In fact, several subjects in each group thought the purpose of the cuff induced pain was

to distract them in their efforts to produce alpha rather than to measure their tolerance changes. Only two of the subjects correctly determined the purpose of the experiment.

The effectiveness of suggestion in pain relief has been shown by Hardy et. al. [1952], Barber [1959 & 1971], Barber and Hahn [1962], and Melzack [1972] to be of major importance in the mediation of pain. For this study, it was the experimenters intent to examine the effect of alpha control alone in the relief of experimentally induced pain.

Hypothesis 7 is not supported by the results of the present study. The results indicated no significant difference in pain tolerance between those subjects classified, for purposes of this study, as "High Alpha" producers and those classified as "Low Alpha" producers, when the feedback tone was present. However, it is important to note that with Hypothesis 8, when no feedback tone was present, the High Alpha producers displayed a significantly greater tolerance to pain than the Low Alpha producers. The lack of any significant difference between the two groups performance when the feedback tone was present may have been due to the very presence of the tone, which afforded both groups an external, attention focusing stimulus. The subjects were instructed to direct their attention to the feedback tone, use it as a guide, and keep it on as long as possible. Focusing one's attention elsewhere has been shown by Chertok [1959] and Sternbach [1968] to have a considerable analgesic effect. It is likely that the tone's presence acted in an attention

focusing capacity for both groups and consequently equalized tolerance performances between the two groups.

With respect to Hypothesis 8, the fact that there was a significant difference in pain tolerance between the two groups when no tone was present is thought to be interesting in light of reports of persistent alpha activity in the EEGs of some individuals demonstrating elevated pain tolerance levels [Chertok, 1959; Anand et. al. 1969; Lawrence, 1972]. It offers some support to the contention that the generation of alpha brainwaves is a contributing factor in mediating pain. The relaxation that accompanies the alpha state, as Melzack [1972] hypothesizes, may produce a general decrease in arousal inputs, as well as a decrease in anxiety, which contributes to the relief of pain.

The ability to maintain a relaxed mind and body has been shown to be an extremely effective aid in coping with the associated pains of childbirth and other non-natal, painful stimuli [Chertok, 1959; Fehmi, 1969; Anand et. al. 1969; Lawrence, 1972; and Melzack, 1972]. Although the results of this study do not provide conclusive results that alpha training has any significant mediating effects on pain, the experimenters believe the study has advanced the need for further investigation to clarify the specific effects, if any, of alpha brainwaves on the pain response. Particularly deserving of further research is the significant difference found in the pain tolerance of High Alpha producers versus Low Alpha producers under no feedback conditions.

C. ANXIETY REDUCTION (PHYSIOLOGICAL INDICES)

For Hypothesis 9, the finding that there was no significant difference between the alpha and yoked groups' ses-sional reduction of systolic blood pressure coincides with the findings of Hypotheses 1 and 2 that there was no significant difference between these two groups' alpha enhancement. It is important to note that both the alpha group and the yoked group differed significantly from the beta contingent group both in alpha enhancement and systolic blood pressure reduction. These results indicate that subjects who receive relevant alpha reinforcement (alpha group) and intermittent alpha reinforcement (yoke group) perform better in reducing their systolic blood pressure than subjects who receive relevant reinforcement contingent upon their beta brainwave activity.

Both the alpha and yoke groups were more successful at enhancing their alpha activity than the beta group. As previously reported by different studies [Kamiya, 1969; Melzack, 1972; Lawrence, 1972; Brown, 1974], there is a strong correlation between the generation of alpha brain-waves and relaxation. The experimenters believe that the difference in the groups' systolic blood pressure reduction may have resulted from the different successes achieved in enhancing alpha activity. From the subjects' verbal reports following each experimental session, it was noted that the majority of the subjects in the alpha and yoke groups expressed

a feeling of relaxation. They associated some form of mental relaxation and body relaxation with the alpha enhancement tasks. Several of the subjects in the beta group expressed a fidgetiness and frustration. One beta subject described how he produced alpha as, "I had the most success holding alpha on by creating a tense feeling in my head as I do when driving at night in a storm." Brown [1974] points out that the psychologic state has been found to be an important correlate of the blood pressure level, and that anxiety and agitation have been significantly related to elevated pressures. The alpha and yoked groups were involved in training tasks which lead to body tranquility. Conversely, the beta group was involved in a brainwave training task that led to a state which was, apparently, less relaxed than the other groups.

Hypothesis 10 is not supported by the results of the present study. The results indicated no significant difference in the sessional reduction of diastolic blood pressure for subjects given relevant reinforcement (alpha group), intermittent reinforcement (yoke group), or beta contingent reinforcement (beta group).

As previously indicated, due to equipment limitations, blood pressures could not be continuously monitored, but had to be measured using standard medical examining techniques at the beginning and end of each session. This could possibly have been the reason that there was found a significant difference in the groups' systolic blood pressure

reduction but not in the groups' diastolic reduction. No evidence on this point is available at present, and the specific effects of alpha brainwave feedback on systolic and diastolic blood pressure reduction needs to be further investigated.

With respect to Hypothesis 11, the finding that there was no significant difference between the alpha and yoked groups' sessional reduction in pulse rate also coincides with the findings of Hypotheses 1 and 2, that there was no significant differences between these two groups alpha enhancement. However, note that both the alpha and yoke groups differed significantly from the beta contingent group in alpha enhancement, systolic blood pressure reduction, and now pulse rate reduction. These results indicated that subjects who receive relevant alpha reinforcement (alpha group) and intermittent alpha reinforcement (yoke group) perform better reducing their pulse rate than subjects who receive relevant beta contingent reinforcement. The experimenters believe that this difference in pulse rate reduction was a result of the different degrees of success achieved in alpha enhancement, and the concomitant relaxation associated with alpha activity, as previously referred to in the discussion on systolic blood pressure reduction.

V. SUGGESTIONS FOR FURTHER STUDIES

In order to express a firm conclusion about alpha feedback and its relationship to pain and anxiety, it is suggested that this research be continued with the following recommendations:

1. For physiological indications of anxiety reduction; blood pressure, pulse rate, galvanic skin response, and muscle tension should be continuously monitored. For psychological indications of anxiety; the Taylor Manifest Anxiety Scale, the Multiple Affect Adjective Checklist, or a comparable test should be used.
2. EEG data should be displayed on the dynograph output along with heart rate, blood pressure, GSR and EMG data to facilitate direct comparisons.
3. Several more training sessions should be conducted to increase subject proficiency at alpha enhancement and control. This should result in a more legitimate test of the relationship between alpha brainwaves and their mediating effects on pain and anxiety.
4. Replicate the experiment, incorporating suggestion to one of the groups that the generation of alpha will effectively diminish pain.
5. Use a larger sample size and a double blind experimental design in order to gain more confidence in the results of the study.

6. Conduct a case study on one subject who has a low tolerance to pain and a high anxiety level.

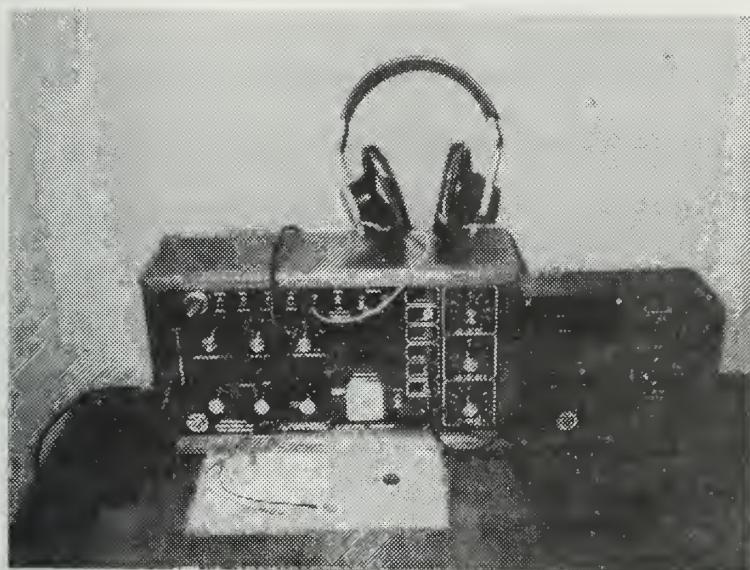
7. The study should be replicated using visual feedback to determine the efficacy of that form of feedback with respect to alpha training and pain mediation.

8. To aid in consistent electrode placement and subject comfort, a helmet or some pre-configured electrode headgear should be utilized.

9. The use of hypnosis to reduce alpha blocking should be investigated.

APPENDIX A

Apparatus



Brain Wave Analyzer
and Aquarius Timer

Subject Seated in
Experimental Chamber with
Electrodes Attached,
Pressure Cuff Applied,
Dynamometer in Hand



Appendix B

Instructions to Subjects

You have volunteered to participate in a study being conducted at the Naval Postgraduate School which involves alpha control and people's reaction to an uncomfortable stimulation. You will be involved in seven sessions of alpha control training, each of which will last approximately one hour. A tolerance measure to an uncomfortable stimulation will be made on the first, fifth and seventh session of your training.

You may have already read about biofeedback research and possibly have your own ideas about an experiment of this nature. Whatever knowledge concerning this area you have is fine, but you are asked not to discuss your ideas or notions about alpha control training with other volunteers. You are further requested not to discuss whatever subjective experiences you may have, resulting from your involvement as a subject in this study. At the completion of your seventh session you will be given a complete explanation concerning the purpose of this study and any questions you may have regarding alpha control and biofeedback will be answered.

While in the alpha training sessions you will have electrodes attached to your head. These are contact electrodes which merely read your brain waves but are of no possible harm to you. During these sessions you are asked to sit as still as possible and relax as much as you can without falling

asleep. At different times in the training sessions the experimenter will ask you, via the intercom, to produce alpha activity or to turn off alpha activity. These tasks will be attempted under two conditions: 1) where you will be receiving feedback, and 2) where you will not be receiving feedback. The feedback mentioned here will consist of a tone received through a headphone set which will indicate to you when you are producing alpha. Remember, the tone indicates the presence of alpha activity and no tone, a non-alpha activity state. This tone can be adjusted for volume and tone, and the experimenter will ask you to indicate a volume/tone preference in the familiarization session. The experimenter will also indicate via intercom whether he wants your eyes opened or closed.

When your task is to produce alpha with feedback, you will be instructed to keep alpha on as much as possible, using the contingent feedback tone as your guide. Recognize the subjective experience during the alpha producing state and try to hold this state when the tone is on. When your task is to produce alpha without a feedback tone, you will be instructed to try to produce alpha on the basis of whatever subjective understanding you might have about alpha. When your task is to achieve a non-alpha producing state with feedback, you will be instructed to keep alpha off using the contingent feedback tone again as a guide. Recognize the subjective experience during the non-alpha producing state of mind and try to hold this state when the tone is off.

And lastly, when your task is to achieve a non-alpha producing state without the feedback tone, you will be instructed to keep alpha off, on the basis of whatever understanding you have about alpha. Are there any questions at this time?

Although, during these alpha training sessions, you are being asked to minimize body movement, it may become necessary to readjust your position from time to time. Please feel free to do so. The experimenter only asks that when you must move, get yourself readjusted comfortably and then to remain still. An occasional movement is much better than continuous fidgeting for accurate brain wave measurements.

As previously mentioned, a tolerance measure to an uncomfortable stimulation will be made on your first, fifth and seventh session. For this measure you will be seated in a sound attenuated room with the electrodes attached. You will be given a hand dynamometer and asked to decide on a comfortable grip setting. This grip setting will be recorded by the experimenter and this same setting will be used on subsequent measuring sessions.

A standard adult-size blood pressure cuff will then be applied to your upper arm and inflated to a pressure level that will restrict the flow of blood in your arm. You will then squeeze the hand dynamometer 20 times. You are to make each squeeze come up to the 10 kilogram mark on the dynamometer. The exercise schedule to follow will be presented to you by tape recorded signals consisting of "squeeze", "hold" and "release."

You will then be instructed to wait awhile to experience and verbally report on the feeling in your forearm. The sensations and discomfort you may experience are due to the temporary lack of blood in your arm. This will cause you no harm whatsoever. You are to verbally report your subjective sensations by using a scale from 0 to 4. Zero indicates a no distress condition, one indicates a slight distress, two indicates when the sensations become moderately distressing, three indicates when the sensations become very distressing and four indicates that point at which you would very much wish to have the cuff removed. You are to verbally call out that number which represents the best description of your sensations as they occur.

In this study we are not interested in evaluating heroics, this is not a test of masculinity nor masochism. We are not comparing tolerance levels between different subjects but are only interested in each participants subjective evaluation of the sensation of discomfort. The experimenter realizes that you can probably endure the discomfort beyond that point at which you would very much wish to have the blood pressure cuff removed. However, we are not interested in that determination and only want an honest subjective report on your part. Is this point clear?

This same procedure of determining a tolerance level to an uncomfortable stimulation will then be repeated for your other arm.

In the fifth and seventh sessions this tolerance level will again be measured but will be conducted while you are involved in an alpha-control training task. When the pressure cuff is on the right arm you will be instructed to verbally report your discomfort as before, while simultaneously trying to maintain alpha on as much as possible using the contingent feedback tone as a guide. When the pressure cuff is on the left arm you will be instructed to verbally report your discomfort while simultaneously trying to produce alpha activity on the basis of whatever understanding you might have about alpha.

After each session, you will be requested to complete a short questionnaire that asks questions pertaining to your physical activities, feelings, and impressions for that day. All information received from such questionnaires or throughout the training sessions will be kept completely confidential and only used in evaluation of your alpha-training.

Thank you for your cooperation. Do you have any questions before we begin?

Appendix C
Training Session Design

<u>FAM</u>	<u>TS-1</u>	<u>TS-2</u>
1. 15 min. BL	1. BL	1. BL
2. PT/RA	2. ON,FB	2. ON,NFB
3. PT/LA	3. ON,NFB	3. ON,FB
	4. OFF,FB	4. OFF,NFB
	5. OFF,NFB	5. OFF,FB
	6. ON,FB	6. ON,NFB
	7. ON,NFB	7. ON,FB
	8. REC	8. REC

<u>TS-3</u>	<u>PT-1</u>	<u>TS-4</u>
1. BL	1. BL	1. BL
2. ON,FB	2. ON,FB	2. OFF,FB
3. OFF,FB	3. ON,NFB	3. ON,FB
4. ON,NFB	4. PT/RA/FB	4. OFF,NFB
5. OFF,NFB	5. PT/LA/NFB	5. ON,NFB
6. ON,FB	6. REC	6. ON,FB
7. ON,NFB		7. ON,NFB
8. REC		8. REC

<u>PT-2</u>
1. BL
2. ON,FB
3. ON,NFB
4. PT/RA/FB
5. PT/LA/NFB
6. REC

Appendix D

Table I

Group Alpha Performances

Training Session One

Group	BL	ON,FB	ON,NFB	OFF,FB	OFF,NFB
Alpha	25.9%	27.2%	29.7%	20.1%	26.3%
Yoke	45.9	45.9	51.2	37.2	43.8
Beta	34.4	20.2	26.3	22.9	23.5

Training Session Two

Group	BL	ON,FB	ON,NFB	OFF,FB	OFF,NFB
Alpha	36.7	37.9	41.7	27.3	25.5
Yoke	30.7	29.1	33.7	27.9	32.9
Beta	40.3	26.8	26.7	29.4	27.4

Training Session Three

Group	BL	ON,FB	ON,NFB	OFF,FB	OFF,NFB
Alpha	23.6	36.7	36.1	19.1	14.8
Yoke	42.4	44.5	42.7	38.9	45.2
Beta	37.1	28.4	28.1	29.0	32.9

Pain Tolerance One

Group	BL	ON,FB	ON,NFB	OFF,FB	OFF,NFB
Alpha	39.8	37.4	41.4	not measured	
Yoke	49.5	50.4	51.0	this	
Beta	42.1	40.1	43.5	session	

Training Session Four

Group	BL	ON,FB	ON,NFB	OFF,FB	OFF,NFB
Alpha	31.5	40.1	37.8	26.9	29.5
Yoke	45.8	47.1	48.4	40.0	42.3
Beta	37.8	35.6	32.5	30.8	34.9

Pain Tolerance Two

Group	BL	ON,FB	ON,NFB	OFF,FB	OFF,NFB
Alpha	39.4	47.2	47.5	not measured	
Yoke	44.9	49.3	43.4	this	
Beta	36.9	36.0	37.2	session	

Table II
Group Pain Tolerance Performance

Familiarization Session

Group	PT/RA/FB	PT/LA/NFB	*Alpha %	
			PT/RA	PT/LA
Aloha	366 secs	322 secs	24.6%	32.2%
Yoke	308	356	31.3	32.9
Beta	303	330	28.7	29.0

Pain Tolerance One

Group	PT/RA/FB	PT/LA/NFB	*Alpha %	
			PT/RA	PT/LA
Alpha	463 secs	404 secs	45.4%	45.3%
Yoke	356	332	38.4	44.8
Beta	323	326	32.6	34.8

Pain Tolerance Two

Group	PT/RA/FB	PT/LA/NFB	*Alpha %	
			PT/RA	PT/LA
Alpha	566 secs	464 secs	51.0%	47.9%
Yoke	402	442	48.6	46.2
Beta	404	311	31.3	37.0

* Indicates % of alpha during pain tolerance measurement

Table III
Group Physiological Measures

Training Session One

Group	BPB	BPA	PB	PA
Alpha	108/76	106/74	64	63
Yoke	115/73	110/72	69	64
Beta	102/69	103/71	66	70

Training Session Two

Group	BPB	BPA	PB	PA
Alpha	104/74	100/71	64	61
Yoke	116/74	114/73	68	65
Beta	107/75	108/79	66	68

Training Session Three

Group	BPB	BPA	PB	PA
Alpha	113/69	106/72	70	65
Yoke	120/72	114/71	69	65
Beta	111/74	116/76	71	68

Training Session Four

Group	BPB	BPA	PB	PA
Alpha	119/70	106/65	67	62
Yoke	123/74	113/72	71	67
Beta	114/76	117/76	68	66

Table IV
 Results Data
 Familiarization Sessions

Subject	BL	PT/RA	PT/LA	*Alpha %		#	PA	BPB	BPA
				PT/RA	PT/LA				
Alpha Group									
1	33.8%	402	646	24.4%	26.8%	62	62	104/58	98/63
2	30.9	531	339	26.0	30.0	80	82	126/84	124/88
3	29.1	140	118	16.1	23.2	66	60	94/62	108/64
4	31.3	402	197	24.2	30.0	66	62	120/74	120/82
5	56.1	355	310	32.1	50.8	64	68	110/68	100/64
Yoke Group									
1	49.3	454	595	28.4	29.0	80	84	116/70	112/74
2	52.5	331	360	43.2	48.0	68	66	90/66	100/76
3	34.7	132	127	34.7	28.9	66	76	128/88	132/88
4	20.7	147	145	13.6	19.6	64	62	104/72	106/70
5	44.5	474	553	36.5	38.8	74	68	114/56	102/56
Beta Group									
1	54.3	181	181	40.4	38.9	78	80	114/68	100/62
2	50.2	166	159	27.3	26.6	64	70	108/64	118/74
3	39.3	413	505	25.6	24.0	74	80	104/76	104/72
4	29.1	604	671	27.4	35.1	92	84	126/90	138/96
5	26.2	151	134	23.0	20.4	50	52	110/62	114/68

* Indicates % of alpha during pain tolerance measures

Indicates 'Pulse Before' the session and 'Pulse After' the session

\$ Indicates 'Blood Pressure Before' the session and 'Blood Pressure After' the session

Training Session One

Subject	BL	ON,FB	ON,NFB	OFF,FB	OFF,NFB	PB	PA	BPB	RPA
Alpha Group									
1	25.6%	33.6%	35.2%	29.8%	31.0%	62	58	94/64	94/58
2	27.0	27.5	30.0	21.9	24.4	80	72	126/104	124/88
3	26.8	24.6	28.7	24.5	35.8	52	60	94/64	94/64
4	32.2	20.1	28.5	19.9	34.9	66	68	120/80	108/80
5	18.3	30.0	25.9	4.6	5.2	60	58	108/70	112/80
Yoke Group									
1	61.0	61.8	70.0	34.4	47.5	86	80	118/74	108/66
2	62.6	52.5	65.1	53.5	60.3	62	56	102/76	104/78
3	36.5	34.5	36.1	33.8	35.0	72	70	132/80	130/84
4	30.4	33.8	36.7	18.7	30.7	52	52	126/70	116/70
5	39.1	46.9	48.2	45.8	45.4	74	62	98/66	94/64
Beta Group									
1	43.4	32.3	42.3	35.5	41.8	78	78	98/66	99/68
2	55.6	16.1	28.3	29.5	38.4	74	76	92/70	100/78
3	17.7	17.1	19.8	13.9	11.1	60	72	96/70	96/70
4	29.8	10.4	14.5	9.5	11.1	72	72	118/80	118/88
5	25.4	25.3	26.7	26.1	25.0	48	50	108/60	104/50

Training Session Two

Subject	BL	ON,FB	ON,NFB	OFF,FB	OFF,NFB	PB	PA	BPB	BPA
Alpha Group									
1	42.6%	38.0%	43.2%	43.2%	32.6%	62	60	104/64	94/60
2	38.7	45.4	42.6	33.4	25.3	80	72	120/90	122/92
3	42.3	46.9	55.6	44.2	48.7	56	58	100/70	86/62
4	31.0	26.9	29.8	11.7	9.3	60	54	105/82	104/76
5	28.9	32.2	37.2	4.0	11.4	62	62	93/66	93/64
Yoke Group									
1	22.7	15.7	24.2	16.1	24.1	76	78	118/78	122/70
2	34.5	30.3	28.7	24.4	42.2	56	62	110/84	108/80
3	21.4	30.6	29.4	33.4	31.5	78	70	126/78	128/78
4	28.7	25.0	29.9	18.8	25.1	52	48	108/70	108/72
5	46.0	44.1	56.3	46.8	41.6	76	66	116/62	102/64
Beta Group									
1	49.8	54.8	46.0	54.0	44.3	80	80	102/74	102/74
2	61.0	14.8	14.9	28.0	34.0	66	66	94/68	86/69
3	41.3	30.4	33.4	27.1	24.4	62	70	118/84	118/88
4	23.0	11.2	13.0	11.8	10.5	72	72	118/84	128/88
5	26.4	22.8	26.3	26.2	23.8	50	52	106/66	106/76

Training Session Three

Subject	BL	ON,FB	ON,NFB	OFF,FB	OFF,NFB	PB	PA	BPB	BPA
Alpha Group									
1	41.4%	38.9%	36.7%	42.6%	32.4%	62	62	118/62	104/62
2	27.2	49.0	41.0	23.6	27.3	82	82	128/76	120/84
3	Equipment Malfunction					70	60	104/72	98/68
4	30.1	26.7	27.6	6.4	4.9	66	56	117/76	110/76
5	20.8	32.2	39.0	3.7	9.4	68	64	100/58	98/68
Yoke Group									
1	40.7	49.3	47.2	43.5	43.8	80	70	120/70	106/70
2	54.9	38.7	49.4	40.7	54.0	72	64	112/80	110/78
3	34.4	36.3	37.8	32.1	34.9	80	76	130/70	130/70
4	42.7	47.6	38.2	35.5	54.5	48	48	118/76	112/78
5	39.2	50.7	40.8	42.7	38.9	64	64	118/64	110/60
Beta Group									
1	36.8	49.3	53.1	58.4	56.7	80	72	106/76	108/70
2	51.7	20.0	29.8	15.6	30.3	70	72	94/70	104/78
3	34.0	38.4	31.2	44.0	44.5	66	69	126/78	126/78
4	38.0	22.1	17.7	18.2	20.4	80	80	120/90	122/88
5	25.1	12.4	8.7	8.8	12.4	58	48	110/58	118/64

Pain Tolerance One

Alpha %

Subject	BL	ON,FB	ON,NFB	RA/FB	LA/NFB	PT/RA	PT/LA	PB	PA	BPB	BPA
Alpha Group											
1	37.2%	32.7%	36.1%	37.5%	40.1%	465	703	62	60	98/56	102/60
2	35.2	35.7	41.9	39.2	36.3	446	243	88	84	140/98	132/94
3	36.4	40.8	43.2	57.0	57.2	113	160	60	62	98/64	96/64
4	34.3	32.0	29.4	35.0	36.1	597	370	72	66	120/64	120/76
5	55.9	45.6	56.3	58.2	56.8	696	543	54	54	110/78	108/72
Yoke Group											
1	53.6	53.7	57.6	34.6	49.0	152	292	82	78	112/68	116/70
2	62.2	55.7	55.4	49.7	59.5	706	497	64	66	118/72	116/80
3	32.7	35.7	37.3	32.5	31.0	124	124	78	72	140/74	132/74
4	41.3	34.9	43.5	23.4	21.8	174	156	52	48	120/76	122/80
5	58.0	71.8	61.4	52.0	62.6	623	593	68	56	108/62	102/68
Beta Group											
1	54.4	56.0	53.3	44.0	46.2	248	167	79	79	108/76	102/70
2	57.9	52.9	65.6	18.2	20.2	171	173	80	70	106/70	110/70
3	45.0	46.0	48.4	35.5	44.5	518	554	60	54	122/82	108/74
4	27.7	31.4	26.6	44.0	43.0	519	599	82	82	128/80	128/86
5	25.5	14.3	23.6	21.5	20.1	158	138	50	50	118/72	122/66

Training Session Four

Subject	BL	ON,FB	ON,NFB	OFF,FB	OFF,NFB	PB	PA	BPB	BPA
Alpha Group									
1	37.3%	35.1%	34.1%	35.4%	33.3%	60	50	107/62	100/60
2	27.5	53.4	40.7	22.4	23.3	80	76	134/86	122/80
3	45.7	41.5	49.5	23.3	39.4	64	64	108/72	98/62
4	26.5	33.0	28.2	24.8	23.9	70	60	128/68	104/64
5	20.7	37.5	36.3	28.9	27.5	60	60	116/60	106/58
Yoke Group									
1	41.1	38.9	38.7	40.1	40.4	92	84	120/72	106/68
2	62.3	69.9	67.4	53.0	56.2	72	68	120/80	116/80
3	29.9	32.9	32.3	28.3	26.8	72	74	142/80	124/72
4	40.1	37.0	42.5	32.7	30.4	52	48	126/80	112/80
5	55.7	56.6	61.0	46.1	57.9	68	64	106/56	106/62
Beta Group									
1	49.6	48.7	48.5	53.1	49.6	84	72	98/66	102/64
2	43.8	42.2	38.1	11.7	35.3	62	66	116/78	108/72
3	41.9	50.7	37.6	62.6	47.3	64	58	112/80	114/84
4	32.3	22.2	25.1	16.3	32.8	84	80	132/86	136/90
5	21.3	14.0	13.1	10.3	9.6	46	51	114/68	126/68

Pain Tolerance Two

		Alpha %									
Subject	BL	ON,FB	ON,NFB	RA/FB	LA/NFB	PT/RA	PT/LA	PB	PA	BPB	BPA
Alpha Group											
1	35.1%	40.2%	38.4%	45.6%	33.8%	788	840	60	60	100/60	100/50
2	35.4	55.0	55.7	49.4	52.6	369	257	82	78	126/80	118/82
3	48.6	48.8	55.9	68.9	64.5	302	354	72	66	94/58	90/52
4	30.3	28.8	28.3	31.4	32.4	470	275	62	58	120/68	120/66
5	47.8	63.3	59.3	59.6	56.1	900	592	52	48	106/76	106/74
Yoke Group											
1	51.4	48.0	49.9	48.6	46.9	611	744	76	78	112/66	108/70
2	59.4	75.8	61.9	68.3	66.7	484	600	78	78	118/72	115/70
3	35.4	32.1	31.2	32.2	30.4	180	136	76	70	140/68	132/72
4	33.2	29.0	21.6	40.1	33.2	154	175	56	54	134/64	126/80
5	45.5	61.8	52.4	53.9	54.3	581	553	60	56	104/62	104/62
Beta Group											
1	34.2	25.7	32.4	19.6	35.6	225	181	78	72	94/64	100/70
2	54.2	60.1	60.8	47.8	44.7	255	227	72	60	114/70	110/72
3	40.5	41.1	41.1	32.3	37.8	636	492	56	52	118/78	116/76
4	29.7	32.7	29.4	38.1	45.9	525	495	86	80	138/98	138/98
5	25.9	20.6	22.1	18.9	20.9	380	158	54	50	128/62	122/54

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